Über dieses Buch

Dies ist ein digitales Exemplar eines Buches, das seit Generationen in den Regalen der Bibliotheken aufbewahrt wurde, bevor es von Google im Rahmen eines Projekts, mit dem die Bücher dieser Welt online verfügbar gemacht werden sollen, sorgfältig gescannt wurde.

Das Buch hat das Urheberrecht überdauert und kann nun öffentlich zugänglich gemacht werden. Ein öffentlich zugängliches Buch ist ein Buch, das niemals Urheberrechten unterlag oder bei dem die Schutzfrist des Urheberrechts abgelaufen ist. Ob ein Buch öffentlich zugänglich ist, kann von Land zu Land unterschiedlich sein. Öffentlich zugängliche Bücher sind unser Tor zur Vergangenheit und stellen ein geschichtliches, kulturelles und wissenschaftliches Vermögen dar, das häufig nur schwierig zu entdecken ist.

Gebrauchsspuren, Anmerkungen und andere Randbemerkungen, die im Originalband enthalten sind, finden sich auch in dieser Datei – eine Erinnerung an die lange Reise, die das Buch vom Verleger zu einer Bibliothek und weiter zu Ihnen hinter sich gebracht hat.

Nutzungsrichtlinien

Google ist stolz, mit Bibliotheken in partnerschaftlicher Zusammenarbeit öffentlich zugängliches Material zu digitalisieren und einer breiten Masse zugänglich zu machen. Öffentlich zugängliche Bücher gehören der Öffentlichkeit, und wir sind nur ihre Hüter. Nichtsdestotrotz ist diese Arbeit kostspielig. Um diese Ressource weiterhin zur Verfügung stellen zu können, haben wir Schritte unternommen, um den Missbrauch durch kommerzielle Parteien zu verhindern. Dazu gehören technische Einschränkungen für automatisierte Abfragen.

Wir bitten Sie um Einhaltung folgender Richtlinien:

+ *Nutzung der Dateien zu nichtkommerziellen Zwecken* Wir haben Google Buchsuche für Endanwender konzipiert und möchten, dass Sie diese Dateien nur für persönliche, nichtkommerzielle Zwecke verwenden.

+ *Keine automatisierten Abfragen* Senden Sie keine automatisierten Abfragen irgendwelcher Art an das Google-System. Wenn Sie Recherchen über maschinelle Übersetzung, optische Zeichenerkennung oder andere Bereiche durchführen, in denen der Zugang zu Text in großen Mengen nützlich ist, wenden Sie sich bitte an uns. Wir fördern die Nutzung des öffentlich zugänglichen Materials für diese Zwecke und können Ihnen unter Umständen helfen.

+ *Beibehaltung von Google-Markenelementen* Das "Wasserzeichen" von Google, das Sie in jeder Datei finden, ist wichtig zur Information über dieses Projekt und hilft den Anwendern weiteres Material über Google Buchsuche zu finden. Bitte entfernen Sie das Wasserzeichen nicht.

+ *Bewegen Sie sich innerhalb der Legalität* Unabhängig von Ihrem Verwendungszweck müssen Sie sich Ihrer Verantwortung bewusst sein, sicherzustellen, dass Ihre Nutzung legal ist. Gehen Sie nicht davon aus, dass ein Buch, das nach unserem Dafürhalten für Nutzer in den USA öffentlich zugänglich ist, auch für Nutzer in anderen Ländern öffentlich zugänglich ist. Ob ein Buch noch dem Urheberrecht unterliegt, ist von Land zu Land verschieden. Wir können keine Beratung leisten, ob eine bestimmte Nutzung eines bestimmten Buches gesetzlich zulässig ist. Gehen Sie nicht davon aus, dass das Erscheinen eines Buchs in Google Buchsuche bedeutet, dass es in jeder Form und überall auf der Welt verwendet werden kann. Eine Urheberrechtsverletzung kann schwerwiegende Folgen haben.

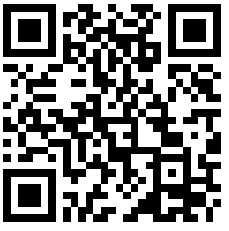
Über Google Buchsuche

Das Ziel von Google besteht darin, die weltweiten Informationen zu organisieren und allgemein nutzbar und zugänglich zu machen. Google Buchsuche hilft Lesern dabei, die Bücher dieser Welt zu entdecken, und unterstützt Autoren und Verleger dabei, neue Zielgruppen zu erreichen. Den gesamten Buchtext können Sie im Internet unter `http://books.google.com` durchsuchen.

This is a reproduction of a library book that was digitized by Google as part of an ongoing effort to preserve the information in books and make it universally accessible.

Google™ books

https://books.google.com

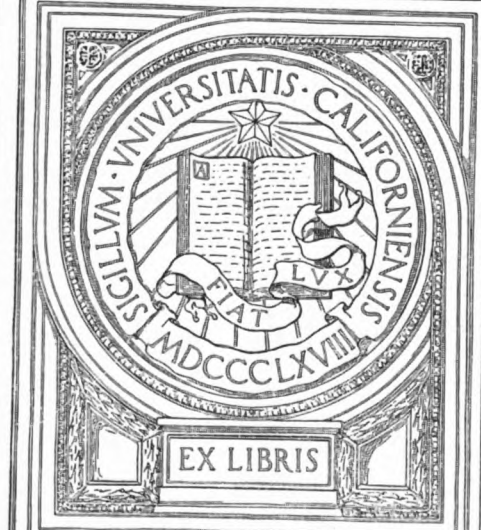


Clinical journal

North Shore University Hospital. Medical Staff

Digitized by Google

UNIVERSITY OF CALIFORNIA
MEDICAL CENTER LIBRARY
SAN FRANCISCO



Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

THE

CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

A WEEKLY RECORD OF CLINICAL MEDICINE AND SURGERY, WITH THEIR SPECIAL BRANCHES.

IN TWO VOLUMES ANNUALLY.

VOL. XVIII.

APRIL 24—OCTOBER 16, 1901.

NINTH YEAR.

EDITED BY

L. ELIOT CREASY, M.R.C.S.ENG., L.R.C.P.LOND.

THE MEDICAL PUBLISHING COMPANY, LIMITED,
22½ BARTHOLOMEW CLOSE, LONDON, E.C.

MCMI.

Digitized by Google

Digitized by Google

LIST OF CONTRIBUTORS.

PAGE

ABRAHAM, PHINEAS S., M.D., B.Sc., F.R.C.S.I. Demonstration of Cases of Skin Diseases ... 392

ALLBUTT, T. CLIFFORD, M.D., F.R.C.P., F.R.S. Clinical Lecture on "Some Neuroses" ... 225

BALLANCE, C. A., M.S. At St. Thomas's Hospital 64

BATTEN, F. E., M.A., M.D., M.R.C.P. Clinical Lecture on Cerebral Diplegia in Children ... 33

BEAUMONT, W. M., M.R.C.S. Clinical Remarks on Four Cases of Intra-ocular Hæmorrhage 335

BEEVOR, Sir HUGH, Bart., M.D., F.R.C.P. Two Cases of General Venous Engorgement ... 14

BENNETT, Sir WILLIAM, K.C.V.O., F.R.C.S. Clinical Lecture on Certain Complications occurring after Operations for the Cure of Hernia 241

" " Clinical Lecture on some Points connected with Thrombosis 401

BERRY, JAMES, F.R.C.S. Surgical Consultation at Medical Graduates' College and Polyclinic 278

BLUMFELD, J., M.D.Cantab. Some Remarks on the After-effects of Anæsthetics 252

BOYD, STANLEY, F.R.C.S. At the Hospital for Consumption and Diseases of the Chest, Brompton 145

BRISCOE, J. F., M.R.C.S. The Practice of Bloodletting 76, 90, 174, 299

BRUCE, J. MITCHELL, M.A., LL.D., M.D., F.R.C.P. Clinical Lecture on the Diagnosis, Prognosis, and Treatment of Quiescent Tuberculosis 209

BRUNTON, Sir LAUDER, M.D., F.R.S. Clinical Lecture on the Medicinal Treatment of Disease 6

BRYANT, J. H., M.D., F.R.C.P. Some Remarks on the Causation and Diagnosis of Acute Intestinal Obstruction 113, 136

" " Clinical Lecture on a Case of Muscular Atrophy... ... 327

BUTLIN, H. T., F.R.C.S., D.C.L. Clinical Lecture on the Surgical Treatment of Tuberculous Glandular Affections in the Neck 321

CHEYNE, W. WATSON, F.R.C.S., F.R.S. At King's College Hospital 49

COLLIER, H. STANSFIELD, F.R.C.S. At the Hospital for Sick Children, Great Ormond Street 315

DUCKWORTH, Sir DYCE, M.D., LL.D., F.R.C.P. Clinical Lecture on Chorea or Cerebral Rheumatism 236

" " Clinical Lecture on the Incidence, Nature, and Treatment of Chorea 369

ECCLES, W. MCADAM, M.S., F.R.C.S. Two Clinical Lectures on the Control of Arterial Hæmorrhage and the Treatment of Aneurysm 348, 363

EDEN, T. W., M.D. Gonorrhœa in Women 342

FISHER, THEODORE, M.D., M.R.C.P. Two Cases of Swelling of the Eyelid occurring after a Bath 318

FLEMMING, PERCY, F.R.C.S. The Ophthalmoscopic Changes met with in Bright's Disease ... 109

FREYER, P. J., M.D., M.Ch. Demonstration of Cases at the Medical Graduates' College and Polyclinic 177

Digitized by Google

PAGE

GALLOWAY, J., M.A., M.D., F.R.C.P. In the Skin Department of Charing Cross Hospital 410
HAWTHORNE, C. O., M.D., M.R.C.P. Clinical Demonstration at the Medical Graduates' College and Polyclinic 214
HENSLEY, P. J., M.A., M.D., F.R.C.P. Clinical Lecture 353
HUTCHINSON, JONATHAN, F.R.C.S., F.R.S. Clinical Demonstration at the Medical Graduates' College and Polyclinic 15, 126
" " Clinical Lecture on Raynaud's Phenomena ... 305
KEETLEY, C. B., F.R.C.S. Two Clinical Lectures on Intestinal Ulceration 247, 265
KELLOCK, THOMAS H., M.A., F.R.C.S. Clinical Lecture on Nævi and their Treatment ... 17
KELYNACK, T. N., M.D., M.R.C.P. Arsenical Poisoning from Beer Drinking 222
LANE, W. ARBUTHNOT, M.S. Clinical Lecture on Movable Kidney 97
" " On the Treatment of Foul Wounds 199
" " Mechanical or Traumatic Arthritis 358, 377
LATHAM, ARTHUR, M.A., M.B.Oxon., M.R.C.P. Clinical Lecture on Cardiac Pain and its Treatment 396
LEES, D. B., M.D.Cantab., F.R.C.P. At the Hospital for Sick Children 385
LOCKWOOD, C. B., F.R.C.S. Cardinal Rules in Diagnosis 172
MORTON, CHARLES A., F.R.C.S. Clinical Lecture on a Case of Recovery from Fracture Dislocation in the Upper Dorsal Region 372
MOULLIN, C. MANSELL, M.D.Oxon., F.R.C.S. Clinical Remarks upon the Treatment of Thrombosed Varicose Veins of the Leg ... 239
PAKES, W. C. C., D.P.H. A Case of Acute Glanders 112
PITTS, BERNARD, M.A., M.C., F.R.C.S. Demonstration of Surgical Cases at the Hospital for Sick Children 405
POWER, D'ARCY, F.R.C.S. Intussusception and its Treatment 268
ROBERTS, FREDERICK, M.D., F.R.C.P. A Practical Outline of "Case-taking," with Comments 257
ROUGHTON, E. W., B.S., F.R.C.S. Surgical Consultation at the Medical Graduates' College and Polyclinic 294
ROUTH, AMAND, M.D., F.R.C.P. The Diagnosis of an Ovarian Cyst 65
" " Clinical Lecture on the Chorea of Pregnancy 193
SHEILD, A. MARMADUKE, M.B., F.R.C.S. Clinical Lecture on Hydrocele 129
SHENTON, E. W. H., M.R.C.S., L.R.C.P. A Note on the Diagnostic Value of X-ray Photography in Osteo-arthritis 85
SMITH, E. NOBLE, F.R.C.S.Edin. Spring Finger 31
SMITH, PRIESTLEY, F.R.C.S. Clinical Lecture on Strabismus 337
SYERS, H. W., M.A., M.D.Cantab. Clinical Lecture 27
TUBBY, A. H., M.S., F.R.C.S. Four Clinical Lectures on some Affections of the Kidney 86, 122, 157, 187
TURNER, W. ALDREN, M.D., F.R.C.P. Clinical Lecture on some Cases of Tabes Dorsalis 71
WALSHAM, HUGH, M.A., M.D., M.R.C.P. Present-day Skiagraphy 152, 161, 184, 203
WASHBOURN, J. W., M.D., F.R.C.P. Clinical Lecture on South African Dysentery 289
WHITE, W. HALE, M.D., F.R.C.P. In the Wards of Guy's Hospital 81, 104
" " Clinical Lecture on Tubercular Peritonitis 273
WINDLE, J. D., M.D. Remarks on the Summer Diarrhœa of Infants 191

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 443. WEDNESDAY, APRIL 24, 1901. Vol. XVIII. No. 1.

CONTENTS.

PAGE

*The Clinical and Pathological Relations of the Chronic Rheumatic and Rheumatoid Affections to Acute Infective Rheumatism. By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P. ... 1

*A Clinical Lecture on the Medicinal Treatment of Disease. By SIR LAUDER BRUNTON, M.D., F.R.S. 6

*With Sir Hugh Beevor at King's College Hospital. TWO CASES OF GENERAL VENOUS ENGORGEMENT ... 14

*A Clinical Demonstration at the Medical Graduates' College and Polyclinic. By JONATHAN HUTCHINSON, F.R.C.S., F.R.S. 15

Specially reported for the Clinical Journal. Revised by the Author.

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 1s. 6d. each, or will be forwarded post free on receipt of 1s. 9d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

THE CLINICAL AND PATHOLOGICAL RELATIONS OF THE CHRONIC RHEUMATIC AND RHEUMATOID AFFECTIONS TO ACUTE INFECTIVE RHEUMATISM.*

By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Physician and Lecturer on Medicine at St. Bartholomew's Hospital.

MR. PRESIDENT AND GENTLEMEN.—I have tried to study the problem implied in the text of this address from various points of view from my student days onwards, and some of my earlier and later essays were amongst the results of this.† But he must be a bold man who, in this first year of the twentieth century, sits down to attempt to find a new niche in nosology for the condition recognised as rheumatoid arthritis. The equipment of a student in the sixties of the last century is a sadly inadequate one wherewith to face the requirements of a pathological leader in the first decade of this era. The progress in physiology, animal chemistry, and in bacteriology has been so rapid and overwhelming that no practising physician can pretend to have kept pace with it. I can therefore only claim to have advanced from the purely clinical side of this question, to have endeavoured through many years to determine the exact nature of the phenomena presented, and to discover their relation to other forms of disease which present likenesses and analogies to it. We all recognise the difficulty of the subject, and, no less, the confusion that still exists in many minds respecting the varieties of arthritis which lead to deformities. We find that the profession, almost equally with the public, is content to call almost any pain in the body or the joints, even when not obviously inflammatory, by the term "rheumatic,"

* Delivered before the Chelsea Clinical Society, March 19th, 1901.

† 'Proceedings of the Abernethian Society, St. Bart.'s Hosp.,' 1863; address before the Belfast Meeting, Brit. Med. Assoc., 1883; 'Heath's Dict. of Pract. Surg.,' vol. i, 1886; 'Liverpool Med.-Chir. Journ.,' July, 1891.

Digitized by Google

so that the latter word has now become nearly synonymous with pain. To determine exactly the true nature of pain demands an amount of knowledge which can only be attained by one, who with large knowledge of all medicine, takes the time and the trouble necessary for the inquiry. I will begin by affirming that by far the greater number of "pains" we meet with are not truly rheumatic, and own a different pathogeny. Increasing knowledge and exactness compel us to believe that this is the case. In the next place, I will express the opinion that the term "rheumatic gout," applied to cases of rheumatoid arthritis, so far as it is used to signify a blended condition of rheumatism and gout, is one which has probably little justification in pathology, and should only be employed by us to denote the cases in question that distinctly admit of this combination.

To clear the way for a more accurate conception of the conditions recognised as true rheumatoid or osteo-arthritis, it appears necessary to state concisely what we know respecting true rheumatism and its influence on joints, and also what we know of the effects of gouty influences on these structures. Time would fail to describe in detail all the points which demand consideration in order to differentiate these two morbid states, and it is therefore necessary to limit our considerations mainly to the conditions of the joints. In thus contracting our points of view, I am well aware that we lose sight of many facts and concomitants, physiognomical, clinical, and others, which are of prime and essential importance in instituting an exact diagnosis. Before entering upon the details, it appears no less imperative to direct attention to the constitutional habit of body which is concerned, as I believe, in most, if not in all, cases in which we meet with manifestations of chronic arthritis. The doctrines of the diatheses are not much recognised in these days, and few in authority in this country now venture to teach or write about them. They are, I think, commonly believed to be of no value, and remain buried in old books, regarded as mediæval lucubrations of pedantic physicians, who groped in the dark days before microscopes and more precise clinical methods were introduced. Such is not my view, for I have learned to satisfy myself of the truth and importance of some of these doctrines for the best interests of clinical medicine. I find myself therefore in accord with the teaching of the French school in respect of a specific diathetic habit of body, which is specially vulnerable to such peccant matters as generate rhematism, gout, and some other ailments, and prone to react specifically, more or less severely, in respect of the component structures of the joints. We find, therefore, that a large number of persons are thus texturally impressed, and that their tissues present, as it were, a soil in which the specific irritant toxins of these disorders flourish readily and provoke their several manifestations.

Per contra, we find a large number of persons who are not so texturally impressed, whose tissues afford no special nidus for these toxins, and who, though exposed to the same conditions, manifest no indications of these maladies. We meet with a precisely analogous condition in respect of proneness to tuberculosis, and textural unfitness or inability to encourage it. Such pregnant facts relating to the tissue peculiarities of the human race surely demand our recognition and study, and we have therefore to realise that some persons are thus immune, and can never become rheumatic, gouty, or tuberculous, however much exposed to such influences. I am disposed, then, to predicate for the majority of persons who suffer from the various disorders we are considering a basic diathetic proclivity towards them, a line of textural or tissue vulnerability, constituting a weak point and malign tendency in their special constitution, which cannot therefore be regarded as normal or sound.

With respect, then, to rheumatic fever, which we have now to include amongst the specific infective disorders, we find that a simple and ordinary attack leaves, as a rule, nothing behind it in the several affected joints. Indeed, this may be the case after repeated attacks. The arthritis subsides, and has no disabling sequels. The same is, happily, sometimes, but far less often, the case in regard to the heart and its component tissues. Too often, indeed, we have to face the results of various degrees of endocarditis, pericarditis, and myocarditis. We are not now concerned, however, with the questions of visceral rheumatism or visceral gout. Gout when it alights upon the joints induces arthritis in varying degrees with less or more deformity as a sequel. Deposition of sodium urate in the articular cartilages, synovial

Digitized by Google

fluid, and even in the bone, is commonly met with, and is regarded as the touchstone of gout by many, though I do not myself hold that view, having found that true gout may not always leave uratic deposits permanently behind it. They may be sometimes removed in course of time. The deformities of gouty arthritis are due to gross disorganisation of the component tissues, to indolent effusions of synovia, to ecchondroses and exostoses, and in the upper limbs, more especially at times, to uratic deposits of large size in adjacent tendons and their sheaths, and in the integuments. The exostoses are rather specially significant in severe cases of gouty arthritis, and give rise to the worst pains, especially at the periphery of the joints, where they form lipping outgrowths. Nodular joints are thus formed, especially affecting the phalanges and their tubercles. The terminal phalanges of the fingers may be thus affected in the gouty, giving rise to one of the varieties of Heberden's nodes. Heberden himself denied the gouty origin of these, but this view is disproved by *post-mortem* research, which has certainly shown the presence of uratic deposit in some of them. There are, however, gouty nodes of this variety which contain no urates, and I regard them as gouty because they occur in association with other plain manifestations of gout, although they may never have been the seats of classical uratic arthritis; and yet they closely resemble those met with in rheumatoid arthritis.

Many truly gouty deformities of joints are, by themselves, indistinguishable from those induced by rheumatoid arthritis. This is especially the case in persons of feeble constitution, the subjects of atonic gout, and particularly in scrofulous subjects who develop gout. The diagnosis here is extremely difficult at times, and can only be arrived at by a study of the family history and the concomitants of the case. Mistakes are only too easily made, and occasionally an exact diagnosis is impossible. I omit, of course, all reference to abarticular or visceral gout.

I lay stress on the fact of exostoses in true gout, and on that which is even more noteworthy, respecting the occurrence of true ankylosis or bony synostosis in this disease, a condition which we do not find in rheumatoid arthritis. Eburnation of the bone may also be met with in gouty arthritis. The opinion is held by some authorities that the uratic deposit follows a preceding degeneration in the textures of a joint, and never occurs in normal tissues in any part of the body. Absolute proof of this is not however forthcoming, and we must beware of making definite pronouncements on the evidence alone afforded in the deadhouse, where we usually see the worst outcome of disease. Urates have certainly been found in articular cartilage which, when deprived of them by solution, appeared histologically quite normal.

It is noteworthy that museum specimens of gouty deforming arthritis, incrusted with urates, if preserved in *methylated* spirit, speedily lose their uratic deposits, whereas if they be kept in pure alcohol they undergo no change. Thus we lately lost a large series of specimens at St. Bartholomew's Hospital collected by Dr. Norman Moore, while we were able to show perfect ones which had been put up seventy years previously in pure spirit. The spoiled specimens, it was interesting to observe, manifested many of the features of rheumatoid arthritis, and might well have passed as such, had no history been attached to them. The fact must then be accepted that gout may, independently of uratic deposit, induce the varied changes, or most of them, which are generally accepted as due to rheumatoid arthritis, and I must here confess that I have found reason to alter the opinion on this point which I formerly expressed to the contrary.

We take note of the special vulnerability of the joints in gouty persons, of the tendency for injuries and strain of these structures to be amongst the determining causes for the alighting of gout in these parts, and we recognise the lingering quality pertaining to arthritis in the gouty as a consequence of their specific habit of body. An analogous, but different susceptibility pertains to persons of strumous habit. The joints thus injured are *loci minoris resistentiæ*.

Attention has been called by Dr. Hawthorne to the occurrence of subcutaneous nodules in rheumatoid arthritis, and, in a careful study of the relations of this disorder,* he brings forward sufficient proofs of the fact. My own experience does not enable me to agree with him as to the frequency of their occurrence, but the fact must be accepted, and it naturally furnishes a connecting link with

* 'Rheumatism, Rheumatoid Arthritis, and Subcutaneous Nodules,' Churchill, London, 1900.

Digitized by Google

so-called true rheumatism. I can also add that nodules of a similar nature occur in some cases of unequivocal gouty character, being met with in the sheaths of tendons, persistent, and void of urates. In both cases the nodules are apt to be persistent, as is the case commonly in adults. The nodules met with in acute rheumatism in the young are more apt to be ephemeral. I am strongly disposed to regard the occurrence of nodules, even without a history of rheumatic or gouty antecedents, as indicative of the so-called arthritic habit of body. Again, there are to be met with instances of gouty, non-uratic changes, often very painful, in the tendons and their sheaths, which last for many months, and commonly subside, leaving no crippling as a sequel. Once more, the condition known as Dupuytren's contraction of the palmar fascia is, in my experience, exclusively the appanage of the gouty or goutily disposed, and is unconnected with true rheumatism. I should not fail to mention here the fact that persons of truly gouty habit are by far more than others vulnerable to the worst effects of gonorrhœal poison in any degree of intensity.

We have next to consider the morbid anatomy of what may rightly be termed chronic rheumatism. By this I mean the sequels in the joints arising out of cases of acute infective rheumatism. In these instances the primary disorder fails to pass away, and a lingering arthritis remains. Hence we meet with enlargement of certain joints, stiffness, pains, and some degree of crippling. In many of these instances I feel sure that the disorder has alighted on a strumous soil, and that this blended diathesis is responsible for the result. There is a special tendency here to indolent effusions in the joints, and they are rebellious to treatment. The general health is enfeebled, and efforts for repair are slow and ineffectual. Many of these cases are regarded as examples of rheumatoid arthritis, and the exact diagnosis is not always easy, inasmuch as infective rheumatism may and does sometimes open the way for later developments of rheumatoid arthritis, and, as we have just seen, the same lingering and deforming arthritis may eventuate after attacks of true articular gout, which own no specific rheumatic antecedents. We are here in face of the question, which demands a reply, as to the possibility of a blended condition sometimes of true infective rheumatism and true gout. It appears to be difficult to deny that such a conjunction is conceivable, but to establish the fact great care would be necessary to ascertain the antecedents and concomitants in any given case, and hardly anything short of an ultimate autopsy could fully justify an opinion. It is well ascertained that in cases of rheumatoid arthritis there is often to be found a history of tuberculosis in near relations.

The variety of rheumatoid arthritis in children associated with glandular and splenic enlargements, described by Dr. Still, I venture to regard as a possible outcome of blended strumous and rheumatic disease.

A study of the clinical features of rheumatoid arthritis, together with that of its morbid anatomy, indicates a marked variety of conditions. It is clear that the disorder occurs with varying degrees of severity, alighting here lightly, and there heavily. We find it involving juvenile textures, and again as an appanage of senility; now as a solitary arthritis, and now as symmetrical; again as a polyarthritis. Here, in response to local injury, there, starting quietly and making rapid progress towards permanent crippling. Equally certain is it, in my opinion, that some inherent defect of tissue or special proclivity in the individual (arthritic diathesis) lays him open, as a vulnerable subject, to be more or less gravely involved by the morbid process. Careful inquiry commonly reveals some constitutional weakness or frailty in the family history. It may be rheumatic or gouty proclivity more or less strongly inherited, and not seldom do we find a strumous element blended with this. Exposure to unwholesome environment, depressing circumstances, nervous exhaustion, mental care and anxiety, imperfect nutrition or enfeebled powers of disposing of nutriment, are common antecedents of the onset; and certain occupations entailing rapid changes of temperature, with exhaustion, or residence in new or damp uncellared dwellings, in warm moist climates, or on clay or water-logged soils, are recognised amongst fruitful sources of the disorder. To these we may add prolific childbearing, hyperlactation, menorrhagia, and the disorders attending the menopause. These antecedents, I say are, and have long been, included amongst provoking conditions, and the results are clearly and duly assigned to them. The subjects of the disease are just those who are more prone

Digitized by Google

than others to fall victims to any intrusive factors whose nature it is to alight especially upon the articular or locomotory system.

The specificity or unity of rheumatoid arthritis as a malady, is held by some observers to be open to grave doubt. In a recent contribution* Dr. Derwent Parker, after a careful and thoughtful study of the whole subject, has declared that all examples of the disease are solely the outcome, respectively, of true rheumatism or true gout. This doctrine demands careful criticism. Within the last five and twenty years it has been held by thoughtful observers that the special characters of rheumatoid arthritis, that is, its clinical and pathological features, may be set up by various morbid processes, amongst which may be ranked rheumatism and gout, but no less certainly by such toxic conditions as are evolved by gonorrhœa and a modified pyæmia; and in addition we have to reckon the arthritis of bleeders, pneumococcal arthritis, alveolar pyorrhœa, dental caries, the post-influenzal and post-scarlatinal varieties. Again, the influence of strain, overwork, traumatism, and diseases of the spinal cord has to be included amongst the antecedents in predisposed subjects.

As I have often remarked in this respect, as "many roads lead to Rome," so many ætiological factors may lead to the onset and progress of what we term rheumatoid arthritis. There can be little doubt that the various elements of the morbid anatomy of this malady may be encountered in joints which are the seat of varieties of arthritis which, during life, have not presented the symptoms ordinarily assigned to rheumatoid arthritis. I have elsewhere recorded my belief that "identity of morbid change is no proof of the identity of the existing cause in any given case,"† and, as I just now remarked, I entertain no doubt as to the fact that gouty arthritis is capable of inducing most of the changes that are to be witnessed in a well-marked example of rheumatoid arthritis. *But the two conditions are not identical.* The process leading up to the one is very different from that which induces the other. I leave out the factor of uratic deposition for the moment, since I do not believe that the arthritis in its grosser degree is solely determined by such deposit, for the coarser osteal, cartilaginous, and synovial alterations are far in excess of any process that could be thus excited, and we know, further, that there may sometimes be a copious but quiet and painless deposit of uratic salt in joints without any gross or exuberant arthritis. I have already mentioned some of the specific changes met with in gouty arthritis which are proper to it, and are never encountered in rheumatoid arthritis, to wit, synostoses which, of course, entail true ankylosis. Again, we find that it is not a common event for true rheumatism to lead to the specific disorder we recognise as rheumatoid arthritis, and thus my experience refutes the dogmatic opinions expressed on these points by Dr. Derwent Parker.

We are therefore compelled to fall back upon the clinical features of the disorder in any given case where morbid anatomy does not tell the whole story, for it is acknowledged by careful and experienced pathologists that many of the gross changes observed in various forms of chronic arthritis are indistinguishable from those presented by unequivocal examples of rheumatoid arthritis. And it is not difficult to understand that such must be the case. All the component textures are involved in a more or less chronic and irritative process, and they can but react in a similar fashion. The specific irritants in the series are gradually being relegated to their respective causes, as diplococci in rheumatic arthritis, gonococci in gonorrhœal arthritis, pneumococci in pneumococcal arthritis,* and hæmorrhage in hæmophilic arthritis. In regard to uratosis as the sole and direct irritant in gouty arthritis, the question remains open whether the gross changes often met with are exclusively dependent on this. I do not believe that urichæmia comprehends the whole pathogeny of gout.

* 'St. Bart.'s Hosp. Reports,' vol. xxxvi, 1900, p. 177.
† 'Treatise on Gout,' p. 56, 1898.

* "Pneumococcal Arthritis," Dr. Cave, 'Lancet,' Jan. 12th, 1901.

(To be concluded.)

We have received from Messrs. Callard and Co., 65, Regent Street, manufacturers of starchless and sugarless foods, some samples of dietary articles for the diabetic. The claim that these diabetic foods are free from sugar is proved by experiment and experience. They can be confidently recommended in the treatment of diabetes and obesity.

Digitized by Google

A CLINICAL LECTURE

ON

THE MEDICINAL TREATMENT OF DISEASE.

Delivered at the Medical Graduates' College and Polyclinic, Chenies Street, April 10th, 1901,

By SIR LAUDER BRUNTON, M.D., F.R.S.

GENTLEMEN,—I confess that until within ten days ago I did not know myself what the subject of my lecture to-day was to be. But I happened to get a pamphlet sent to me from the Polyclinic announcing that this lecture had been fixed for to-day, and that the subject of it was to be "The Medical Treatment of Disease."

The most extraordinary lecture I ever listened to was one given by the late Professor Balfour, of Edinburgh. In this lecture he discussed the whole subject of botany, beginning with the vegetable cell, passing through the natural orders, and finishing up with the geographical distribution of plants and fossil botany—all in the space of one hour. I do not think I can attempt any feat of the sort, and if I did attempt it I should certainly not succeed. And yet you may think that I am attempting very little else in this lecture, having as its subject "The Medical Treatment of Disease." But I do not intend to give you the whole medical treatment of disease, because it would be perfectly impossible to do so in the time at my disposal; all that I intend to do is to give you some thoughts about the general relationship of the treatment of disease by drugs to the treatment of disease by other methods, and to insist upon one or two little practical points that I think are sometimes not sufficiently attended to.

You all know that some years ago there was a phase of medical scepticism, during which it was rather the fashion for some medical men to say, "*I* do not believe in drugs," and to pooh-pooh those who did. But this scepticism was not entirely satisfactory to the patients, because the reason why patients consult us nine times out of ten is that they want us either to relieve or to cure the diseases from which they are suffering, and if we cannot do this, they think it is not much good having us in.

Though unsatisfactory in itself, this scepticism was not without its good side, for it was the reaction from the meddlesome medicine of a previous period. Oscillation or revolution, which may be regarded as a form of oscillation, is the movement *par excellence* of all things in this universe, whether they be molecules, worlds, or phases of thought. A reformer rarely succeeds in removing an abuse unless he goes to the other extreme, and medical scepticism was an almost inevitable step from mischievous meddling based on ignorance and fancy to successful treatment based on real knowledge. You all know the comparison of a doctor to a policeman who interferes between a burglar and the citizen whom he is attacking. The well-intentioned policeman draws his truncheon, and hits hard at somebody. In the dim light he sometimes hits the citizen, sometimes the burglar, and it is a mere matter of chance whether he does harm or good. Disease is the burglar, the patient is the citizen, and the doctor the policeman who, by using powerful measures, may sometimes put a stop to the disease, but may sometimes, alas! hurt or even kill the patient.

In the dim light of empirical knowledge there can be no doubt that the physician sometimes did much harm, and the excessive use of mercury and blood-letting hurt or killed many a patient. But the light of increasing knowledge lessens day by day the risk of hurting the patient, and increases the likelihood of destroying the disease. Witness the effects of antiseptics. Antiseptics almost without exception are harmful, and in sufficient quantity destructive not only to the microbes which produce disease, but to the delicate protoplasm of the cells which compose the various tissues and organs of the human body. And yet not only the medical profession, but the public at large knows how diseases due to microbes may be prevented, controlled, or cured in a way that is little short of miraculous. Compare the feelings of people in a large city now with what they were three and a half centuries ago when a case of plague was said to have occurred. Then there was panic and terror; now there is no doubt some anxiety, but it is relieved by the knowledge that we have not to fight without weapons against an intangible pestilence, but against an invasion of microbes which we can certainly destroy by antiseptics if we only can get them applied fully and freely enough. Even when pathogenic microbes are not destroyed by antiseptics, they may be so much weakened as

Digitized by Google

to have no power to resist the attacks made upon them by the living cells of the tissues in our bodies. For the struggle for existence between the organism and pathogenic organisms is a real fight, and may be very fairly compared to one between a citizen and a burglar. Returning to this illustration, we can readily see that if the policeman even without hurting the burglar can distract his attention, and give the citizen time to rest and draw breath, he will aid him greatly in the struggle; and if he could give him a pull at a brandy flask, so much the better. Now in the struggle of the organism with disease, it must be the physician's great aim to maintain the strength of the organism by rest, food, and stimulants, quite as much as to attack the disease. The *vis medicatrix naturæ* is no empty phrase. Nature unaided may do much, but Nature aided by judicious regimen and proper medicines will do much more. But care must always be taken that the regimen and drugs are judicious, and are not calculated to do more harm than good.

Now, bodily disease is very much like national or social disturbances. You know that nations are subject to disturbances from within, civil wars, strikes, party dissensions, etc., and to invasions from without; and the same thing takes place in our bodies. Sometimes disturbance in the body arises from the want of co-ordination between the action of different parts, and sometimes it arises from the invasion of microbes from without. The first instance that I know of in which this want of relationship between the different parts of the body as a cause of disease is mentioned, is in the fables of Æsop. In this fable of "The Belly and the Members"* it is said that the Members took some offence at the conduct of the Belly, they called it fat and indolent, and resolved no longer to grant it the usual supplies. The Hands protested that they would not lift a Finger to keep him from starving; and the Teeth refused to chew a single morsel more for his use. But in a very short time the Members found that the Stomach was not only a highly respectable organ, but it was indispensable to their welfare, and ought to be treated accordingly. Now, we find that the usual cause of strikes is overwork, or at least that people want to work less than they do. This is very much what occurs in the body. Organs strike work because they want to do rather less than they are doing; they are as it were "put upon," and they want rest, either comparative or absolute, for a time. I do not know that there is any organ more long-suffering than the stomach, but sometimes it is treated with so little respect that it goes on strike, and then the consequences for the rest of the body are very bad. Nor do I wonder that it sometimes does go on strike; the wonder is that it does not do so oftener. I have seen a man stick his fork into a new potato bigger than a pigeon's egg, give one gulp, and down it went; and then the man said that new potatoes were indigestible. If he had simply taken the trouble to give his teeth a fair share of the work, he would not have been troubled with indigestion. But the stomach is not a gizzard, and it will not be treated as such. If it is treated thus it is apt to strike, and one of the chief occupations of any doctor who sees much of digestive disturbances, is to insist upon the necessity of rest for the stomach by giving the teeth a larger share of the work, and making the patient eat slowly and masticate his food thoroughly. At the same time we can sometimes ease the stomach by giving less food or more digestible food, for many people eat too much, and many are too careless as to what they eat. We find also that if the stomach is weak we can give it assistance, either by helping secretion or by helping its movements. We increase its power of secretion by stimulating it before meals with a little bicarbonate of soda, perhaps along with a bitter tonic, and we give after meals a certain quantity of digestive enzymes, such as pepsin or something of that sort. But I think that very often we lose sight of the fact that secretion of the stomach—the gastric juice—will act more rapidly if you do not dilute it too much, and that it is necessary to take care that the digestive enzymes are not too much diluted. If a patient drinks much water during a meal it is evident that the gastric juice becomes diluted, and therefore digestion goes on more slowly. But the point to which I wish to draw your attention just now is this:—many patients have been told by their doctors not to drink during a meal, but they think that they may drink as much as they like immediately afterwards. But if they drink immediately after a meal they dilute their gastric juice very much

* This fable was spoken by Menenius Agrippa, a Roman consul and general.

Digitized by Google

as if they drank during the meal. They ought not to be allowed to drink until a couple of hours or more after the meal, until, in fact, digestion is nearly complete and the stomach is ready to empty its contents into the duodenum, and then they may take as much water as they like. But here comes another point: if the stomach is feeble in its movements it may find a difficulty in emptying itself, and in atonic dyspepsia you may possibly find that, along with general weakness of the body, the stomach itself is feeble, and hangs down much lower than it ought to, so that instead of the food getting out of the stomach along a nearly horizontal line, it has to be hoisted up almost in a vertical direction. In order to lessen this difficulty you may give your patient directions to lie down after food for a certain length of time, say half an hour to an hour, or you may aid the stomach by putting on a fairly tight band. I have found a great deal of trouble in getting bands to fit the abdomen, and for this reason I frequently recommend, instead of a band made by a surgical instrument-maker, a simple strip of flannel half a yard broad, and doubled, so as to make a band nine inches in width, long enough to go one and a half times round the abdomen, and fastened with two or three safety pins so tightly as to give full support to the abdomen. In this way you tend to help the stomach mechanically, whilst at the same time you may stimulate its muscular fibres by the use of strychnine alone or with acids such as nitro-hydrochloric; and it is a significant fact that strychnine is one of the drugs most commonly employed in the practice of medicine.

But it is not merely rest to the stomach which we want to bring about; we frequently want to prevent the effects of invasion from without, the bad effects of microbes. Microbes in a healthy stomach have not much chance; they are soon destroyed by the gastric juice; but if the secretion of this juice be feeble, its antiseptic power is weakened, and the microbes may grow and thrive. In order to destroy them we give various antiseptics. But just consider, what is the good of pouring antiseptics in small quantities into a stomach that is full of all sorts of things? You know that in many cases where there is dilatation of the stomach, if the patient were sick he would fill a large basin with the vomit, and if you are going to put one drop of carbolic acid into a basinful of fermented liquid, is that going to stop fermentation? You say at once that it will not. In order to give your antiseptics a fair chance you must wash the stomach out first, get rid of most of your ferments, yeasts and bacteria, and if you then give your antiseptic, it will have a chance of acting.

In the intestine also we must regard both the conditions under which the intestine is placed and the drugs that we put into it. You know quite well that if we give some irritant before a meal, for example, if we prescribe five minims of liquor arsenicalis before a meal, it is very likely indeed to bring on gastro-intestinal irritation, vomiting, and diarrhœa. If we give the same drug after meals, so that it becomes mixed up with the food, it will very likely have no local action whatever. The same thing holds good with regard to those irritants which we give for the purpose of stimulating the bowels. An old gentleman whom I knew was in the habit of taking a pill every night for at least forty years, and this acted very well. It was a very small pill, only one grain of compound colocynth pill, one of compound rhubarb pill, and one grain of extract of hyoscyamus. This acted with perfect regularity, and he thought the reason was that he gave his pill a fair chance. Instead of putting it into a full stomach he always took it about five minutes before dinner, so as to give the little pill a start of the food and an opportunity to act on the empty stomach. I have very little doubt that this five minutes before dinner made a great deal of difference in the action of his purgative, and that if he had taken the pill five minutes after dinner it would very likely not have had anything like so much effect.

Everybody knows that, however nice a food may be, people get tired of it if they always have it day after day. Partridge is a very nice thing indeed to take, but if you have partridge every day you get tired of it, and many people seem to wonder very much that the intestines should be somewhat like the palate and become tired of a single purgative, so that if it be taken night after night it ceases to have effect. If you tire of partridge you go on to some mutton, and if you tire of mutton you go on to beef. Treat your bowels in the same way as the stomach. If they are tired of cascara go on to rhubarb. If they get

Digitized by Google

tired of rhubarb go on to colocynth, and from colocynth pass on to senna, and so on; and by the time you have gone the round of purgatives the bowels will be ready for the first one again. If you want to soothe the bowels instead of stimulating them, you must of course be careful about the food, and see that there is nothing irritating in it to stir up the bowels mechanically. At the same time you may often find it necessary to give some antiseptics, just as in the case of the stomach, and here again it is well to clear out the bowels first, so as to give the antiseptics or sedatives fair play. I am not going into the question of antiseptics in the intestine. They are very numerous and very useful, but I wish you to remember that we have to treat the bowels like the stomach, and like the rest of the body too, in being careful to remove external irritants, and one very powerful irritant of the intestines is a sudden change of temperature. Many of you may remember some years ago a great discussion about electric belts: and at a trial concerning these belts a number of people said that they had got the very greatest possible benefit from them. I have no doubt they did. It was not because they were electric belts, because I think it was shown that they contained no electricity, but because they were *belts*. And sometimes in cases where you fail to get any good at all from your astringents you may find that diarrhœa may be arrested by the use of a large belt to the abdomen, and more especially by a belt made of chamois leather, made much in the same way as the flannel belts I have already described.

There are certain cases of diarrhœa which are exceedingly troublesome. They will go on for years, and you treat them until both you and the patient are sick of it; sometimes there is chronic continuous diarrhœa, sometimes, and perhaps more frequently, two or three days diarrhœa alternating with two or three days of constipation. The motions frequently contain quantities of mucus, showing a considerable amount of catarrh in the intestine. In such cases you try a number of remedies, but many of them do not succeed. I dare say very few of you have seen tannin used for intestinal complaints; I have very rarely used it for these myself, and the reason is chiefly this: that tannin expends its energy on the stomach, and it has little left to act on the intestine. Therefore, if you are going to give tannin in any form for the intestinal trouble, it must be in a somewhat insoluble form, such as catechu or kino, so that it will act on the bowel. One is afraid to mention a new drug, lest such mention should be taken by the manufacturers, and without leave, to one's great annoyance, as an advertisement. There are some new drugs which give good results in diarrhœa, but I will not mention them by name for the reason I have just given. Amongst the older ones I may mention that small doses of copper sulphate or copper chloride, one third to half a grain, given in a pill, sometimes acts very beneficially. But perhaps there is one old-fashioned drug which acts even better in cases of chronic catarrh of the colon, and that is castor oil. Many of you may know that there was a sort of endemic dysentery in Millbank Dispensary, and that at one time the late Dr. Baly was physician to the Dispensary. He tried all sorts of drugs for this dysentery, and at last he hit upon castor oil, and used it in a mixture containing 10 minims of oil made up with mucilage of acacia and peppermint water. This mixture, if it were not for its abominable taste, is an exceedingly good thing in cases of chronic catarrh of the colon. In some cases I have given a 10-minim dose in a capsule, with one minim or two minims of tincture of opium. Sometimes where these small doses do not seem efficient, we find that a teaspoonful given every morning will do more for the chronic diarrhœa than anything else I know.

In regard to the stomach and intestine we have always to remember that the blood from these organs has to pass through the liver before it ever gets into the general circulation, and that if we are to do much good in a case of congestion of either the stomach or the intestines we must be careful that the circulation through the liver is free. The liver is a very sensitive organ, and is very liable to suffer from chills. I do not know that any organ is more readily affected by chill than the liver without the patient being aware of it. Frequently people come and say "I do not know what I have eaten or drunk that has disagreed with me, but my digestion has gone all wrong." Very often it is not that the patient has eaten or drunk anything which is indigestible, but that he has got a chill to his feet or shins, his liver, or the back of his neck. Most frequently in such cases the person has been

Digitized by Google

sitting in a draught between the door and the fire, and the chill has come about without his knowing it. Here, again, the old-fashioned remedy of five grains of blue pill taken at night and a black draught in the morning sometimes is of great benefit. This remedy is sometimes good even in cases of chronic diarrhœa. I remember very well a man who came from Assam to consult me, and he had been suffering from chronic diarrhœa for several months. His liver was large and congested, and I said to him "there is one remedy I should like to use, but I do not like to give it without consultation. I would like to use sulphate of magnesium and blue pill, but it seems so queer to give a man a purgative who is suffering from chronic diarrhœa." I took him to Sir Joseph Fayrer, and he agreed with the diagnosis and with the treatment. We gave the man a course of blue pill and sulphate of magnesium, and the result was that his liver went down to its normal size and the diarrhœa ceased.

We have been speaking a good deal about the effect of rest to the stomach, but in cases where there is a severe lesion of the mucous membrane, either in the stomach or in the duodenum, we find the advantages of medication. Just now, with changeable weather, I think there has been an unusually large number of cases of gastric ulcer, and this very morning I saw a lady who had been suffering such intense pain that, without stopping to ask a doctor or her husband, she had gone to consult a surgeon with the idea of having her abdomen opened and the ulcer sewn up. It happened that the ulcer was a duodenal one, and a surgical operation for its treatment would have been almost impossible. But in most of those cases you can stop the pain almost to a certainty by the simple use of bicarbonate of soda, only you must use it in large quantities. The best way is to take a big teaspoonful of bicarbonate of soda, dissolve it, and let the patient sip the solution teaspoonful by teaspoonful until the pain is gone. I have said you should dissolve it, but I have nôt mentioned the solvent. Formerly I used to dissolve it in water, but now I almost always use lime water, and add to the lime water some spirit of peppermint. The reason I use lime water now is this. Some years ago I was called to a very severe case of duodenal ulcer, and I prescribed bicarbonate of soda. It acted like a charm as far as relieving the pain was concerned; but a little while afterwards the patient was seized with hæmorrhage, which recurred and recurred, and finally proved fatal. One knows that in severe gastric ulcer, or duodenal ulcer, hæmorrhage may occur of itself, so that I could not say it was due in this case to the bicarbonate of soda that I prescribed; but it occurred to me afterwards that the bicarbonate of soda might possibly have softened the tissues, and thus rendered the patient more liable to hæmorrhage; and ever since I have used lime in some form along with the bicarbonate of soda. Now, in cases of ulcer of the stomach or duodenum you generally find there is a good deal of constipation, so by giving lime you are tending to increase the constipation. To lessen this you may give with the bicarbonate of soda and lime water some fluid magnesia, and thus you will counteract the tendency to constipation at the same time as you neutralise the acid of the gastric contents, and you relieve the pain, sometimes as if by magic. In cases of gastric or duodenal ulcer I think you will find that neutralising the acid of the stomach and duodenum will relieve the pain even when fairly large doses of morphia have failed to subdue it.

Instead of dissolving the bicarbonate of soda in lime water, the following may be used:

℞	Spt. menth. pip.	... ...	ʒiss.
	Cretæ præparat.	... ...	ʒss.
	Magnes. carb. lev.	... ...	ʒj.
	Sodii bicarb....	... ...	ʒj.

M. Ft. pulv.

A teaspoonful to be mixed with half a tumbler of water or more, and slowly sipped, a teaspoonful at a time, till the pain is relieved.

Another part of the intestinal canal that is very apt to be troublesome is the rectum. Piles are a very common condition. Rest here is one of the greatest boons that you can give to the patient. The piles very often are apt to come down after a motion, and if you can manage to get the patient to go to stool in the evening instead of in the morning, it will allow the patient to rest after the process of defæcation, and allow the piles to remain in the rectum after being replaced, instead of coming out again and being subjected to friction as is likely to be the case if the bowels are relieved

Digitized by Google

earlier in the day. There is a tendency in many people for prolapse of the intestine to occur not only through the anus, but also of the sigmoid into the rectum. This condition is not infrequently associated with a feeling of depression and soreness after the bowels have been opened, and here again rest at night immediately after the motion will do a great deal of good. Astringents, both to the piles and to the prolapsed sigmoid, are very beneficial, and the old-fashioned gall and opium ointment to the piles, and an astringent solution injected into the rectum and allowed to remain there in contact with the upper part of the rectum, and with the prolapsed sigmoid, will have a good effect. You may, if you like, introduce gall and opium ointment into the bowel by means of an ointment introducer for prolapse of the sigmoid, or you may not require to use opium, and may simply inject a solution of tincture of catechu, two drachms in two ounces of water, and allow that to remain in the bowel as long as the patient can retain it. But there is one very simple thing which does more to relieve piles than anything else, and that is careful washing immediately after defæcation. However carefully the fæcal matter may be wiped away from the anus in persons who are suffering from piles, a little fæcal matter is very likely to remain between the piles, and act as a constant irritant during the day. If it be carefully washed away immediately after a motion the patient has no further trouble, and the piles are not again extruded, as would be the case if there was a little irritant matter still remaining there. Sometimes you may find that the addition of 1 or 2 per cent. of cocaine to the gall and opium ointment helps its action very considerably. Another drug which is very useful is hamamelis. You may make a mixture of half an ounce of gall and opium ointment, and half an ounce of ointment of hamamelis or similar proprietary preparations, with enough cocaine added to make a 1 per cent. ointment. This gives great relief in many cases of piles.

Another part of the body which is very much tried just now by the changeable weather is the throat and larynx. You find many people suffering at the present time from very severe colds. We all know that the cough which accompanies a cold is due to reflex irritation, and this irritation is generally in the trachea, bronchi, or lungs. But the irritation in these places may not be sufficient in itself to produce cough, yet it will do so if it be supplemented by irritation from somewhere else. And thus we have what is known as stomach cough. Irritation of the stomach does not cause cough, but irritation of the stomach *plus* irritation in the trachea or bronchi may cause it. You may ease such a cough by giving simply bicarbonate of soda in much the same way as I have recommended in cases of gastric ulcer. I have been inclined to think, from my experience within the last few weeks, that there is another additional cause of cough, namely, irritation at the back of the nose. Sometimes you get irritation of the back of the nose with a certain amount of mucus forming there, and a cough which comes on severely, and is not stopped merely by treating the larynx or the trachea, but is lessened by clearing out the mucus from the naso-pharynx. Where there has been irritation of the larynx I have been using parolein a good deal, with 1 to 2 per cent. of cocaine, and 3 or 4 per cent. of menthol. This not only gives good results for the nose and for the back of the throat, but is useful also in cases of irritation in the larynx.

Another remedy by means of which one can stop laryngeal coughs, which have, I think, been more usually common of late, is a spray directed on to the larynx itself. There are various substances as sprays which act perfectly well, but a spray containing 1 per cent. of morphine, or a similar quantity of heroin, which is a new and apparently a useful preparation, or 1 per cent. of cocaine, either alone or combined with morphine, tends to ease the cough for the time being, and to stop the tremendous spasm which comes on. In these cases of throat cough there is very often a certain amount of hoarseness. The cough is exceedingly violent, and nothing seems to stop it, and yet little or no expectoration comes away. If you look into the throat the vocal cords are congested, and perhaps a little thickened, and possibly not meeting so well in the middle as they should do. But there is nothing more to be seen. For these cases the local application is, I think, better than the general use of morphine, which tends rather to interfere with digestion.

In the case of every organ of the body we find that rest is one of the greatest boons when disease is present, and I do not think I have ever

Digitized by Google

done so much good by any drug in cases of cardiac disease as I have done by prescribing rest. It is one of the hardest things in the world to get that prescription carried out, because many of the cases are in men who have been accustomed, not only to work hard themselves all their lives, but to drive everybody else round, and you cannot get it into their heads that they must remain quiet now. There are two things which it is very hard to get men to do: it is very difficult to get a lazy man to work, but it is still harder to keep an energetic man quiet. In cases of cardiac disease rest is, I think, above all things the panacea. But rest alone will not do everything you want, and one must use along with it various remedies. The chief ones are strychnine to stimulate the heart, and digitalin to increase its power; but we must remember in using those drugs that the heart may be somewhat degenerated, the fibres may be somewhat fatty, and if we use these drugs we tend to increase the contractile power of the vessel, and to oppose a greater resistance to the heart. Therefore we may sometimes find that instead of doing good we are doing harm. How are we to meet this difficulty? I think we may meet it to a considerable extent by giving at the same time some drug that will tend to dilate the vessels. And so instead of leaving the patient to himself you may prescribe, along with rest, digitalis or strychnine, and possibly some caffeine also; but combine these with spirit of nitrous ether, with nitroglycerine, or nitro-erythrol. Thus, at the same time that you are causing increased contraction of the heart, you are dilating the vessels, and so in every way accelerating the circulation of the blood. But we must never forget that in cases of heart disease the condition of the blood has very much to do with the cure of the patient, and when the ordinary method of treatment fails entirely one may sometimes succeed by coming back to the old-fashioned plan of giving one grain of blue pill, one grain of powdered digitalis, and one or two grains of powdered squill three times a day. I have had a patient under my care lately, suffering from very bad mitral regurgitation as well as mitral obstruction, with a great deal of œdema, and, other drugs having failed, we have put the girl upon this old-fashioned pill, and continued it longer than one usually does,—so long, in fact, that we began to get the well-known action on the gums. And, curiously, it was just when the action of the mercury began to be manifested by soreness of the gums that the œdema began to distinctly subside. This is an observation that is in accordance with what one reads in the old books: but at the present day we do not, as a rule, push mercury so far. I believe that very often if we were to push our drugs, especially mercury, a little further, in the way our predecessors did seventy years ago, we might sometimes get better results.

Another remedy that has recently been introduced is one which I wish to bring before your attention. Many of you have probably used it largely, and it may not be new to you. I refer to urotropine, as a remedy both in cases of cystitis and also in cases where you wish to disinfect after typhoid fever. Not very long ago Dr. Horton Smith showed that the urine of typhoid patients contained large numbers of typhoid bacilli, so that a new channel of infection was pointed out by him. Before that, one had been accustomed to regard the fæces as being the chief channel of infection; but now we know that urine, either coming accidentally in contact with the utensils or lying upon sheets, may be a means of conveying infection, and if you put such patients upon urotropine you may remove one chance of infection. Therefore now, as a matter of routine, we have been accustomed to give urotropine towards the end of an attack of typhoid fever in order to destroy the infectiveness of the bacilli, these having been found to be present and to be infective after the fever is practically terminated. In cases of catarrh of the bladder there is no drug like it, but I think it is often given in too small doses,—for instance, five to seven grains; whereas, if you want to get the best results, I think you must give it in doses of ten grains or more. When the larger doses are given I have seen no bad results, but I think you may often get distinct advantage with a large dose when you fail with a small one.

The only other point I would draw your attention to is the effect of rest, combined with certain drugs, in nervous exhaustion and brain fag. Everybody is accustomed to have his night's rest in order to prepare for the work of the succeeding day. But many people do not seem to consider that the night's rest is not always sufficient to recuperate from the work of the preceding day, and to render the person properly fit for the work

Digitized by Google

of the succeeding day, and that therefore there is a little residuum of weariness over. In many people this residuum becomes very definite towards the end of the week, so that they feel worn out, and they want one whole day in which to rest themselves. If they do not take that rest they break down in consequence. I do not know that this is so evident in any other class of people as it is amongst clergymen; and the reason that so many clergymen break down is that they do not keep the fourth commandment, "Six days shalt thou labour and do all thou hast to do:" but the clergyman does not do this, and on the seventh day, when he ought to be resting as well as everybody else, he is working hardest. Now, if you have any clergymen to treat, insist that they shall keep the fourth commandment. It is not one day in seven or two in fourteen which will do, but every seventh day; the seventh day must be the day of rest, so that the day of rest is regularly recurring. Clergymen, as a rule, cannot take Saturday, but insist upon their taking Monday if they have to work hard on Sunday. But Moses showed his wisdom in another thing. He knew that even the seventh day was not sufficient sometimes. He knew there might be a residuum, and so he ordained a year of rest every seventh year. The Americans show their wisdom in this. American professors get a year off every seven years, during which period the work of the Professorship is taken by an assistant, while the Professor himself goes away and recuperates, and at the same time learns all that is going on, so that he goes back fresh in mind and knowing a great deal more than he did when he left, and so the students reap the advantage. Now it is very odd that there are so many people who will not take rest, although nature seems to be pressing it upon them. You will find patients coming and telling you that they are always tired, and that they would like to sleep week in and week out. If you tell them to go to bed for a week they will think there is nothing nicer, but they will not do it until the doctor tells them to do so. Then they will go to bed, have a thorough rest, and recover. But rest is merely good in one way, in so far as it allows the tissues time to recover; but sometimes it is bad in another way, in so far that the quietude may tend to allow the products of waste to accumulate, and so occasionally you may not only have to insist upon rest so as to recuperate, but you may have to remove the products of waste by massage. And where other methods have failed you can have recourse to the method introduced by Weir Mitchell and known by his name, of complete rest of body and mind, forced feeding and massage. In the cases which are not quite so bad as this you get somewhat the same result by a long sea voyage, for then the patient is resting, is unable to get much exercise, while the continued motion in the open air gives him an appetite. Moreover he is isolated from telegrams and letters, or intrusion of any kind, and thus there is rest of the mind. But sometimes patients cannot go through the Weir Mitchell treatment or have a long sea voyage, and then you may have recourse to drugs. There is one combination of drugs which I have found very satisfactory, and which I give very frequently, and that is a mixture of bromide of potassium and salicylate of soda. The reason this mixture is so useful is, I think, simply that the bromide quietens the nervous system down, whilst salicylate tends to remove the waste products. If you are going to sweep out a room you have two things to do; you have to apply the besom, and sprinkle water so as to prevent the dust from rising too high. That is what you do in giving salicylate of soda and bromide of potassium; the besom is the salicylate to clear out the waste products, and the bromide of potassium is the damp which quietens the nervous system. Therefore the two combined are exceedingly good, not only in preventing or removing headache, but in preventing what is sometimes of the very greatest trouble to the patient and his friends, irritability of temper. And I feel quite sure that each one of you must require a dose of both of those drugs after having listened to such a long lecture, with a patience for which I cannot thank you too warmly.

Cholelithiasis.—C. W. Mitchell cites a case of cholelithiasis which was operated upon successfully. The material taken from the common duct showed pure culture of typhoid bacilli. He believes we are justified in the statement that the *Bacillus typhosus* produces gall-stones — firstly, because the bacillus is often found in the biliary channels when gall-stones are present; secondly, it can be artificially grown in pure culture when so obtained; and lastly, bacilli so cultivated can produce gall-stones.—*Maryland Med. Journ.*

Digitized by Google

WITH SIR HUGH BEEVOR AT KING'S COLLEGE HOSPITAL.

TWO CASES OF GENERAL VENOUS ENGORGEMENT.

THE Todd Ward now contains two patients with evident general obstruction of the circulation and visible venous engorgement. A comparison of these two cases raises common and important questions in diagnosis.

The first, a woman, age forty-eight, tells a usual tale. After a few years of dyspnœa, recurrent attacks of circulatory incompetence from advanced mitral disease render her unable to move about. In this case the diagnosis proceeds from ample evidence both at the heart and at every periphery of the circulation.

At the heart there is marked right-sided enlargement; evidence of hypertrophy of the right ventricle is given in the sense of impulse palpable to right of the apex. The maximum impulse, however, is very little displaced from the normal. The pulse, it is important to note, before treatment and since, has been at the rate of only seventy to eighty. These two last facts are of every day aid to us in forming the more particular diagnosis as to the form of mitral disease present.

Hearing a systolic murmur at the apex and beyond, and not hearing any diastolic or presystolic murmur there, too often suggests to us to attach undue importance to the regurgitation, and to fail in recognition of a more dominant obstruction of the mitral orifice. So in this case the systolic murmur is most pronounced, the presystolic often absent; but from the former data we must hold that the high pressure in the pulmonary circuit, announced by the accentuated sound over that artery, is in the main owing to narrowing of the valve.

To define the condition of the right side of the heart and the competency of those valves, other evidence again than that of auscultation is of the greatest value. Here the systolic murmur is loudest between apex and sternum, but in the absence of pulsation in the enlarged liver, and of regurgitant pulsation in the veins of the neck, it may be explained by the hypertrophy of the right ventricle allowing a better path for the conduction of sound, just as in a simple case of mitral regurgitation the sound is conveyed well against the blood-current to where the mass and proximity of the left ventricle can approach the ear at the apex.

The second case, a girl, age ten, whom Mr. Burghard asked me to see, is at first sight a replica of the woman. There is the same venous engorgement apparent; in the face it has led to congestion of cheek and nose; though the venules are not so dilated, it has caused a bulbous swelling of the nose. The external jugulars are dilated, and up to a visible valve they fill from below; the liver much enlarged and marked ascites; very slight œdema of ankles; urine high specific gravity and from time to time a trace of albumen; no enlargement of spleen; the lungs behind on either side have a few loose rales. In both patients also the temperature runs persistently one to two degrees below the normal. Here the likeness ends, and the girl differs in her history, in evidence of disease of right lung and pleura, in the absence of evidence of heart disease.

The account of the onset is vague: a somewhat rapid onset eight months ago, puffiness of face, and swelling of abdomen then appearing, with some cough, sweating, and wasting; she is fairly nourished now.

The chest on admission, besides the already mentioned râles at either base, showed more pronounced changes, affecting chiefly the pleura, but also the lung. On the right side in front dulness was marked, most intense against sternum, disappearing at border of axilla and above, getting less to the clavicle; over the right nipple an area the size of a crown gave tubular breathing, not loud but definite; there was crepitation over the dull area and diminished breath-sounds; both the tubular breathing and crepitation disappeared a few days after admission.

So far the simplest explanation of the case would be mitral stenosis with a patch of thickened pleura and congested lung. If so it is an advanced case of stenosis. However, apex-beat and heart-sounds are normal—the first sound is not sharp and valvular, the second pulmonary artery sound is not accentuated—we can neither infer the left ventricle is much underfilled, nor that high tension strains the pulmonary artery. The Röntgen-ray screen does not illustrate any cardiac enlargement. Further, the pulse is not markedly

Digitized by Google

small, and its rate has been persistently 120 or quicker.

It is certainly a canon in diagnosis in such a case of obstructed circulation to accept a single cause of obstruction as an explanation, and not seek multiple causes; but in the puzzle before us we may ask what further possibilities of obstruction can occur. The simplest theory which affords but a single cause is that of a marked primary tricuspid stenosis, though here confirmation should be given by the Röntgen-ray screen.

Exceptionally, we see cases where complete occlusion of superior or of inferior vena cava has occurred, usually with no history of their illness; such cases during life are conspicuous by a festoon of large veins on the surface of the abdomen, called a caput medusæ, and after death usually a mere fibrous band of occlusion is found, and further evidence is wanting as to its mode of origin. Is it possible that such an accident here is falling on both the venæ cavæ?

Again, obstruction of the circulation in the lung will affect the whole systemic circulation, but such must first tell its tale upon the pulmonary artery and upon the right side of the heart. Lung signs are not unconspicuous here, but the evidences of congestion of the lung are upon anatomical grounds consistent with the hypothesis of obstruction of venæ cavæ. There is intercommunication between the systemic bronchial and the pulmonary alveolar circulation in health; in disease it explains the early occurrence of bronchitis (not congestion of the lungs) in cases of mitral disease. Contrariwise high pressure in systemic veins might react upon the pulmonary circulation proper. This case still entails attentive observation of the child.

[Since printing the above the woman has died. Great hypertrophy of right ventricle was found. Great stenosis of mitral valve. There was no tricuspid incompetence, but a stenosis of the tricuspid valve; the presence of this in particular was the more difficult to infer in auscultation from the loudness of the systolic murmur.]

Plantar Reflex in Infancy.—Morse concludes from 254 cases that there is no constant plantar reflex in the first year, and that while it approaches the adult reflex during the second year, it is still inconstant.—*Amer. Journ. Obstet.*

A CLINICAL DEMONSTRATION AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

BY

JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

GENTLEMEN,—The patient, æt. 57, who was examined two years ago, says there was no lump found in the breast then. The first thing noticed was that the nipple was retracted, a definite symptom which our forefathers used to rely on very much. The retraction occurs owing to induration immediately under the nipple, pulling on the lactiferous tubes, which become involved in the contracting mass. I never saw such a typical example as this. It is a funnel right down to the nipple, and the nipple itself has a sulcus all round it. Whenever one sees retraction of the nipple one may be sure there is a central tumour, and not a peripheral tumour. A central tumour in the middle of the gland is the only one which can pull on the nipple. Here is a perfectly simple, rounded, hard mass. You see the gland is quite movable on the chest; there is not the slightest adhesion; there is no implication of the skin around it. The mass is of stony hardness, a little ill-defined, not rounded, and it is pulling a little on all the tissues round it. On examining the arm-pit of this side, we find one very hard mass—perhaps there are two glands adherent together,—very definite indeed, and quite movable. The patient has a central scirrhus of the mamma of the contracting form, and of the hardest kind. It is of comparatively recent origin, so there will be no enlarged glands in the neck yet. The surgery of the case is as clear as possible. It is a tempting case for operation. Every gland in the axilla should be taken away, with all the fat and the mammary tissue from the breast towards the nipple, and including the pectoral fascia. The skin is quite healthy, and we need not sacrifice much of it. She tells us that a sister had cancer of the tongue, of which she died. One should always examine both breasts and both axillæ in these cases. I should not regard this as a case in which there could be any room for difference of opinion. She would be well after the operation in a week or ten days, and her prospect of health for a considerable period afterwards is good.

Here is another breast case. The patient is

Digitized by Google

sixty-five years of age. Here there is no retraction of the nipple, but at the side of the breast there is a dimple which looks as if there were a contractile tumour beneath it, in the periphery of the breast. Without feeling deeply at all, I detect a hard ridge at that spot, and on taking hold of it I easily feel a mass between my finger and thumb. The scirrhus in this case is bigger than in the other case. This shows how little retraction of the nipple is to be relied on as a diagnostic feature. This scirrhus apparently begins in the axillary border of the gland, the middle being quite healthy. It, like the other, is ill-defined in its boundary, and is very hard; it merges into the surrounding tissue. There is no suggestion of fluctuation. The hard forms of scirrhus often contract, and sometimes they wither away altogether. This patient has only known of the growth five weeks, and then she discovered it accidentally. There are quite definite glands in her armpit, but not so movable as in the other case. Recently she says she has felt some pain in the other breast, a pain similar to that which attended the onset of the present tumour. We should, as a matter of routine, examine the root of the neck in all these patients. The lymphatics from the axilla pass up under the clavicle. Here there is no evidence of enlargement of glands at the base of the neck. This patient's mother died of enlargement of the liver, but she does not know whether it was malignant. She tells us she has had no blow on the breast, and no other member of the family has had cancer. The surgery in this case is the same as in the last, but I fear it is a slightly less hopeful case. The glands are not quite so hard, and the patient is of a lymphatic constitution. But the prognosis is hopeful in both cases. I expect that both of them will go four or five years without recurrence. I am now seeing a lady whose breast and axillary glands I removed sixteen years ago for a rapidly-growing tumour. I removed another gland from her armpit, which we discovered three or four months afterwards. But now, at the end of sixteen years of complete freedom, she comes with another growth in the breast, and her glands in the neck are rapidly becoming enlarged. I have had many of these cases, some of which I have lost sight of. One lady who had her breast removed is in her twentieth year of freedom from growth, notwithstanding that it was of the soft variety.

The next case is that of a young woman with a syphilitic ulceration at the back of the tongue, a gumma, and also one on the soft palate. It is a remarkable case. It was a very ugly lesion when she first came, and we cured it with iodide of potassium and mercury, so that there can be no doubt about the diagnosis. She also exhibits thickening of her tibia. She is only eighteen. The question is, Where does the specific disease come in? It is not secondary disease, and the sclerosed condition of the tibia is old. She shows nothing which indicates recent specific infection. There are no indications of inherited syphilis either; the teeth are not affected, nor the iris. The lesion of which she now complains is on the opposite side of the mouth to the original one, a deep ulcerated gumma. It has been thought that probably there has been some accidental inoculation. We do not regard it as syphilis acquired in the ordinary way. As regards inheritance, I might say she is one of a large family, of which she is neither the eldest nor the youngest, and her father and mother and brothers and sisters are all free from any evidence of syphilis. The condition of the tibia is like that found in inherited syphilis, and I have seen many such cases. In acquired syphilis there is a local periostitis, which is sabre-shaped, with the convexity forwards, a term first used by the French, I think. These cases used to gravitate to the Orthopædic Hospital. Cases of syphilitic periostitis used to be under treatment for "bow-leg." Some of you may remember that three or four years ago I showed you a young lady, a private patient, with a large lump like a cherry below and above the tongue,—a hard gumma. In her case I knew the facts. I attended her and her sister for congenital syphilis; but this patient remained well until she was thirty years of age. She has had no relapse. Her physiognomy is not affected, but we know that very often congenital syphilis does not affect the face. The physiognomy and condition of the teeth are a useful guide, but are not to be trusted absolutely. In this patient one of the former ulcers has undergone sclerosis, and is not painful at all. I do not find any symptom of choroiditis, and she says her legs do not ache. She shows the signs of paralysis of one side of the face. That occurred in infancy. Does that help us in the diagnosis? Some neurologists believe that many patients with inherited syphilis get nerve symptoms. Here is a symptom which locates the disease to infancy. Whenever she speaks you can notice the distinct one-sided movements of the face, and she cannot quite shut her eye on the left side. Paralysis of the facial nerve is exceedingly uncommon. I should apply some solution of chinosol, two grains to the ounce, and swab the sore in the mouth with it, and let her take increasing doses of iodide of potassium and sal volatile. This is necessary, because there is not the same rapidity of healing with the iodide of potassium and mercury as before.

BURROUGHS, WELLCOME AND CO. have submitted a convenient tabloid, labelled "Iron Citrate Compound." It is an excellent ferruginous general tonic, containing three grains citrate of iron and ammonia, one grain sulphate of quinine, and one sixtieth grain arsenious acid.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 444. WEDNESDAY, MAY 1, 1901. Vol. XVIII. No. 2.

CONTENTS.

PAGE

***A Clinical Lecture on Nævi and their Treatment,** By THOMAS H. KELLOCK, M.A., F.R.C.S. 17

***The Clinical and Pathological Relations of the Chronic Rheumatic and Rheumatoid Affections to Acute Infective Rheumatism.** By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P. *(concluded)* 23

***A Clinical Lecture.** By H. W. SYERS, M.A., M.D.Cantab. 27

***Spring Finger.** By E. NOBLE SMITH, F.R.C.S.Edin., L.R.C.P.Lond. 31

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

NÆVI AND THEIR TREATMENT.

Delivered at the Hospital for Sick Children, Great Ormond Street, February 28th, 1901,

By THOMAS H. KELLOCK, M.A., F.R.C.S.,

Assistant Surgeon to the Hospital.

GENTLEMEN,—I have chosen "Nævi and their Treatment" as a subject for our consideration to-day at the risk of its being considered rather a minor and trivial complaint, and of being accused of going over again what is well-worn ground; but I think we may sometimes with profit and interest consider some of these minor subjects which acquire importance from the frequency of their occurrence, and in the case before us from the good that can so often be done in relieving a patient of what is always unsightly, and, it may be, a cause of considerable inconvenience, for situate as nævi so often are on exposed parts of the body, it is a matter of no little importance to those who have to earn their livelihood, as well as for appearance sake, that these disfigurements should be as far as possible, and as soon as possible, remedied. As soon as possible, for the earlier in life they are treated the better is the result. How common nævi are may be gathered from the fact that during one year taken at random 83 cases were sent to my out-patient department here alone; this represents one third of the surgical out-patients, so that the total number would be 249 in a year, and this would not include the very trivial ones that would be treated by the house surgeon.

Of the *pathology* of nævi there is comparatively little to say; structurally they are composed of an agglomeration of slender blood-vessels with very little muscular tissue in their walls, lined with the usual endothelium, separated from each other, and at the same time bound together by a small amount of fibrous tissue. The size of these vessels differs in the several varieties, in the superficial ones they are very small, little more than capillaries, whilst

Digitized by Google

in the deeper so-called subcutaneous variety they are often found of considerable size, and in the latter the whole tumour is lobulated and enclosed within a thin capsule of connective tissue, which is not found in those more superficially situated. The circulation of the blood is a little peculiar, arteries lead up to them, and veins carry the blood away, and yet one is often struck in excising them by the fact that the vascularity of the surrounding parts is not very great; cut well wide of one of them, and the hæmorrhage is not much more than if one were cutting into an unaffected part. In the bright red superficial nævi the circulation is rapid, as shown by the way in which the colour returns after they have been pressed upon; and if cut into, or an ulcer penetrates them, they bleed readily, but owing to the thinness of the vessel walls the hæmorrhage is easily controlled by pressure. In the deeper ones the circulation is not so rapid, as shown by their much darker colour; if cut into dark blood issues in a feeble stream, and the hæmorrhage will often cease spontaneously when the vessels of which they are composed have been emptied. That part of the skin which covers a superficial nævus is always badly nourished, in spite of the free circulation underneath, and if ulceration takes place in it from any cause it spreads rapidly, and is healed slowly and with difficulty. The only case I can remember in which death resulted directly from a nævus occurred in this way. The patient was the subject of a very extensive one affecting the whole of one arm and a considerable part of the side of the trunk; ulceration began in the arm, and in spite of all treatment spread to the chest, and the child eventually died of septic poisoning.

Excluding those cases of so-called nævus of the skin of the legs and other parts, which arise in elderly people—women especially—and which are really dilatation of pre-existing veins and capillaries —varicose veins they might more properly be called—and those cases of angioma of the liver which are said to commence in adult life, but which cannot properly be classed with nævi, we may say that nævi are almost without exception congenital; they increase in size, it is true, after birth, rapidly and extensively sometimes, but there is always a something, however small, present at the time of birth. Of their causation we know little or nothing; whether they are hereditary is doubtful, but it is without doubt common to find several children in the same family affected. A maternal impression is often sought as causing them, and you know, perhaps, of a superstition among women which induces them when pregnant if they see anything they think likely to impress a birth or a mother's mark on their progeny to immediately touch themselves on some inconspicuous part of the body in the hope that should the baby be marked it will be so in a corresponding part of its body, and so the disfigurement not be noticeable. It is a curious fact how many more girls than boys are affected by nævi. Out of the 83 cases I mentioned before 57 were girls, and out of 10 cases which were so extensive as to necessitate their admission to the hospital for operation during the year 1900 only one was in a male child. Why this should be so, or what it means, I do not know. It has been stated that children with extensive or numerous nævi are often the subjects of congenital syphilis. I do not think my own experience bears out this statement, and I think those of you who have watched the out-patients here for a little while will agree with me that a large proportion of the babies who have nævi are noticeable for their vigorous health.

Nævi are essentially an affection of the epiblastic layer of the embryo. They are found on almost every part of the skin, and on mucous membranes at the orifices where the epiblast turns in to join the hypoblast, as at the mouth and anus. If they occur in the mucous membrane of the alimentary, respiratory, or urinary tracts, they do not seem ever to be discovered, or to give rise to any symptoms. I should give the following order of the frequency of the parts of the body affected:—trunk, face, scalp, upper extremity, lower extremity. The female external genitals are often affected; not so the penis and scrotum, but this may possibly be accounted for by the relative infrequency of nævi in male children.

Varieties.—Nævi are generally divided into three classes—the superficial, cutaneous, or capillary; the deep, subcutaneous, cavernous, or venous; and the mixed, which are a combination of the other two varieties. I should like, however, to draw your attention to two varieties of the first class which we often see, the diffuse and the circumscribed. The first of these, the diffuse —perhaps better termed a nævoid condition of the

Digitized by Google

skin,—is extremely common in very young children, situate in the upper eyelids, on the forehead in the shape of a V, the apex at the root of the nose, and the base at the junction of the forehead and scalp, and also among the hair immediately under the occiput. If a baby has a nævus on any part of the body you will almost invariably find more or less of this condition in the positions mentioned associated with it; it is a matter more of interest than of any great importance, for the condition needs no treatment, and disappears as the child grows, but it is occasionally of assistance in making a diagnosis when a baby has a subcutaneous tumour of doubtful nature in some other part of the body. Of the circumscribed superficial nævi there are again two kinds, some—the majority—are *flat* hardly rising above the level of the surrounding skin; pass your finger over them, and, except for the sensation of a slight increase in the temperature, you would perhaps hardly recognise any abnormality. The edges of these are often very irregular, and at times their limitation is not very marked. The others are *raised*, almost pedunculated at times, looking like velvet plush cushions; they are always sharply marked off from the surrounding healthy skin, and are very satisfactorily treated by excision for this reason. The deep, subcutaneous, or venous nævi generally form darkish coloured tumours which project but slightly on the surface of the body; certain situations, however, modify this, and they may form projecting or even pedunculated masses.

There is a form of deep nævus which is sometimes associated with tumours and new growths. They are generally termed nævi, but can hardly, I think, be classed pathologically with those found in the skin; they are composed of a collection of thin-walled vessels which are separated from each other by much more, and much looser, connective tissue than we find in the cutaneous variety, and these vessels are, so to speak, in the direct circulation, and not forming a *cul-de-sac*, as in the case of ordinary nævi; in fact, they remind one very much of the condition found in a varicocele, and probably the two conditions have a good deal in common. Of such is the collection of vessels found in those rare cases of congenital unilocular cyst of the axilla, forming a pedicle to this, passing up under the clavicle, and connected with the deeper structures in the lower part of the neck.

Here perhaps is the proper place to remind you that a superficial nævus may be associated with a tumour of a more important kind. It is quite common to find the base of a spinal meningocele or myelocele surrounded by a superficial nævus, and I would warn you to be careful in dealing with a tumour of the back situated in or near the middle line on whose summit you see a nævus; it may, of course, be only a nævus of the mixed variety, but the superficial nævus may also be a very useful danger signal, and the tumour beneath it connected with the spinal meninges or with the spinal cord itself.

Nævi are found too in association with other congenital tumours; it is not uncommon to find them in the skin covering hygromata, or, as in the illustration I show you, in the skin of a limb affected by lymphatic hypertrophy.

Nævo-lipoma is a term one hears used sometimes, but it is a condition one rarely sees. The congenital lipomata have peculiarly few blood-vessels, and it is uncommon to find more than the ordinary subcutaneous fat about a deep nævus.

As to *prognosis*, you may be asked by a parent what will happen to the nævus if it be left alone? This question will probably be asked, not about one on the face or other visible part—these they are always naturally anxious to have removed,—but about those on the trunks or limbs where they are not likely to be seen. Even these, as a rule, mothers are anxious to have removed, for in the first place they seem to have an idea that any disfiguring mark or deformity in their child is a sort of disgrace to themselves, and secondly, they think that should a nævus be cut or knocked the child will bleed to death. I suppose the bright red colour has something to do with the latter idea, which alarms unnecessarily, for, although it is true that if cut, bleeding and rather free bleeding does occur, yet the hæmorrhage can be easily stopped by the mildest pressure; at the same time one must always bear in mind in this, as in every other condition, that loss of blood to any extent is always serious in a young body. A more real danger in nævi of any size is ulceration with sloughing and possibly hæmorrhage as a complication. An injury or continued irritation may start this, and once started there is little tendency for it to cease until almost the whole tumour has been destroyed. I say *almost* because the ulceration often stops

Digitized by Google

near the edge, leaving a ring of nævus next the healthy skin, and this ring, instead of being destroyed by the ulceration, is apt to be, as it were, stimulated in growth, and to spread outwards into previously unaffected skin. If the nævus be large, ulceration taking place in this way may be serious, as in the case I mentioned before, where the child lost her life from septicæmia: on the other hand, if the growth be small and the ulceration extend to its edge, it may result in a cure, but while this is going on there is the danger of sepsis and hæmorrhage, and the cure after all is not a very scientific one. It is possible to imagine malignant disease, sarcoma or epithelioma arising in a nævus. I do not remember ever to have heard of its happening, nor do I know of any recorded case. In addition to these dangers there is always the chance of a nævus spreading, and it is rather remarkable how rapidly this sometimes takes place. A superficial nævus which at birth is hardly visible may attain a very considerable size in a few months or even weeks; more than this a crop of similar growths sometimes springs up around the original one—a good argument in favour of early removal. There seems, however, a limit to their growth, which does not go on indefinitely, but ceases after a time and the tumour becomes stationary. The cause of this cessation of growth is just as obscure as that of its occurrence. The deep nævi very often increase in size after birth, but not nearly to the extent the superficial ones do. These, then, are the risks that are run by leaving a nævus untreated: on the other hand, we must not lose sight of the fact that some of them may spontaneously disappear partially or entirely as the child grows. The nævoid condition about the eyelids, forehead, and occiput always does so, and that too in a relatively short time; look for them when the child is a year old and you will probably find no trace left. The deep cavernous nævi rarely if ever, I think, disappear without treatment, but the diffuse superficial ones sometimes do, and it is not uncommon to watch them getting less bright in colour, although their size may not diminish, and eventually they may become hardly noticeable. How comparatively rare a thing it is to see a child six or seven years old with a nævus. Is it because they are all so effectually treated when they are young? I cannot help thinking that in some of those troublesome cases where from the situation of the growth, treatment is tedious and difficult, that in the eventual curing we are aided to no small degree by a natural tendency of the growth to shrink and disappear as the child grows. This, however, should be no argument against early operation, for we cannot tell whether it will occur or no in the particular case with which we have to deal.

Treatment.—We come now to what is perhaps the most important part of our subject, the treatment of nævi. I have already mentioned those conditions under which no treatment is necessary. For the rest, there are so many ways of dealing with them that perhaps the best plan will be to consider the methods *seriatim*, and under each heading the advantages and disadvantages of the method and the cases for which it is suitable. For consisting as nævi do of several varieties, and situate as they are on so many parts of the body, no one plan can be adopted for them all, and it is necessary to consider for each which of the various methods should be chosen. In the first place we should consider that which has the widest field of applicability, and which is probably the most satisfactory of them all in its results, namely *excision*. This has the advantages of thoroughly removing the growth at one operation. Union by first intention can be obtained, the scar left is the least possible, and becomes practically imperceptible as the child grows, and there is no danger of recurrence if the excision has been properly carried out. It cannot of course be practised in those situations where the necessary drawing together of the skin would cause disfigurement. An anæsthetic is necessary, and the hæmorrhage, even where the greatest care is taken to cut wide of the growth, is often considerable, and not to be too lightly considered in the case of a young baby.

The incisions should be planned so as to be completely clear of the superficial part of the growth, and yet to remove the least possible amount of healthy skin. For the deep ones it is generally possible, by turning back skin flaps, to avoid sacrificing any skin at all. Sometimes two curved incisions, one on either side, sometimes a triangular wound, where the edges are united afterwards in a triradiate fashion, or, again, a quadrilateral wound, united afterwards in "envelope" shape, will best answer our purpose. An infant's skin is wonderfully elastic, and it is remarkable

Digitized by Google

how large a growth may be excised in this way and the edges brought together without undue tension, possibly with the help of a little undercutting of the surrounding parts; even if the edges cannot be brought quite together, the remaining surface can be grafted immediately, and a very good result be obtained; but if this has to be done do not be too free with the suturing, it is better to graft rather more than appears necessary, so that the small amount of contraction that takes place under the grafted surface may not cause disfigurement by dragging on an already tightly sutured part. I always use horsehair sutures in these cases. They are very unirritating, and leave less mark than any others; all the same they should be removed at the earliest possible date, or the little white marks they leave by the side of the scar make the latter more noticeable; a collodion dressing can often with advantage be substituted for the sutures after two or three days. I am referring now, of course, to operations on visible parts of the body; in other parts the marks do not matter much. There are a few situations where excision cannot very well be carried out, where for instance a nævus invades the whole thickness of the lips, cheeks, auricle, or eyelids, in the tongue, on the gums, or where it involves important structures, such as the parotid gland—as in the child I show you to-day. In a female child where a nævus involves the breast, excision would mean the sacrifice of the gland, and should not be done unless every other form of treatment is contra-indicated. A common situation for a mixed nævus is in the scalp over the anterior fontanelle. Here excision is quite suitable, and I think the dangers of interfering with one in that situation have been much exaggerated. They pulsate, it is true, but the pulsation is only communicated, and between them and the superior longitudinal sinus and membranes of the brain there is the thick fibrous membrane closing the fontanelle. I have often excised nævi in this situation, and have met with no more difficulty than with those on other parts of the scalp; but here, again, let me warn you to be careful in making the diagnosis. Subcutaneous tumours at either of the fontanelles at the root of the nose, or at the inner angle of the orbits may be meningoceles or dermoid cysts, both of which communicate generally with the interior of the skull; these tumours are not, I think, so often accompanied by nævi as the similar ones in the back, but the combination might be met with and lead to great difficulty in treatment if their nature had been overlooked.

Where it is not possible to excise the whole of a superficial nævus one may be tempted to excise a part and deal with the rest in one of the other ways. I have not found the results of this plan very satisfactory. I practised it once on the side of the nose, but I think the result would have been better if I had had a little more the courage of my convictions and excised the whole of it and put a skin graft on the raw surface. On one occasion I was about to excise a mixed nævus on the scalp, when I found that leading up to it were two fair-sized arteries lying in grooves in the bones, one on either side of the head. I ligatured both of these a little way from the growth, and for a time the result seemed satisfactory, as the nævus seemed to get smaller and harder; but this benefit did not last, as circulation was re-established, everything returned to its former condition, and I had to excise it. On another occasion I tried to cure a superficial nævus of the chest wall by starvation. I dissected it up, leaving only two small pedicles of skin, one at either end, and then allowed it to fall back, and stitched it in place again. No change took place until later I divided one of the pedicles, and then the nævus began to ulcerate, and this too had to be excised eventually.

Electrolysis is a method of treating nævi of which I am not particularly fond. It is a rather tedious performance, painful, and so needing an anæsthetic probably on many occasions. If, as sometimes happens, sloughing occurs about the spots where the needles were inserted, rather unsightly marks are apt to be left. A large mass of fibrous tissue is often left in the situation of a deep nævus treated in this way, which is in itself a disfigurement, but which can sometimes be subsequently removed by excision. Electrolysis has the advantage that it is bloodless and can be employed in certain parts of the body where other methods of destruction are contra-indicated, where, for example, a nævus involves important structures, such as the deep muscles of the face, cheeks, lips, tongue, eyelids, female breast, etc., and here we can often get improvement at any rate without destroying those structures which are mixed up with the growth,

Digitized by Google

and it is for deep nævi only in these and similar situations that I would use this method. For superficial nævi it is rarely needed; if it destroys them it often does so by causing ulceration, and this can be brought about more thoroughly and quickly by other methods if excision is out of the question. I have often thought, when dealing with a nævus by electrolysis, that part of the improvement was due to breaking up of the vessels composing it by the needles, apart from the coagulation of the blood brought about by the electrolytic action.

In employing electrolysis there is no need for a very strong current; that from eight to twelve cells is generally sufficient, and the current should be allowed to pass through the growth for from five to ten minutes at a sitting. The needles should be insulated to within half an inch of their points, and passed into the growth far enough to prevent any non-insulated part being in contact with the surface, or ulceration is almost sure to occur at the points of insertion. For further details of this method of treatment, I cannot do better than refer you to Dr. Lewis Jones's work on 'Medical Electricity,' where you will find a very complete account of it.

Cauterising agents.—The actual cautery, galvano-cautery, and strong acids are useful in destroying superficial nævi which for some reason cannot be excised. They are often employed too for very minute growths; but I think it is better to excise even these, and for this reason: It is not at all uncommon to find after the destruction of superficial nævi by cauterising, that round their situation there appear leashes of dilated blood-vessels, reminding one very much of those seen in the conjunctiva when there is an ulcer on the edge of the cornea; the same thing is often seen in the situation of the scar of an accidental burn. These vessels will probably disappear again after a time, but they are unsightly if the face happens to be the part operated on. Keloid, too, arises sometimes in the scar left after cauterising a nævus.

An anæsthetic is hardly necessary when cauterising a nævus, especially if it be small. The pain appears to subside very rapidly on the application of a little ointment, or better, dry powder. Whichever of the cauterising agents is employed it must be done thoroughly, or after a few days the scab that has resulted will come off, and the nævus be found as vigorous as ever underneath it. For this reason the acids, nitric or sulphuric, are perhaps better than the cautery, as their effect is more penetrating; but they require more care in their appplication. It is a pitiful thing to see a child's face scarred by acid that has been applied carelessly to a nævus in infancy; to avoid this, when using any kind of caustic the surrounding skin should be protected by vaseline or some simple ointment, and, of course, great care taken that only a very small quantity of the acid is carried by the glass rod or other instrument that is being used. It is hardly necessary for me to add that especial care in this respect should be taken when operating near the eye. A few of the deep nævi which cannot be excised, and which do not respond to treatment by electrolysis, may be dealt with by turning back a flap of skin or mucous membrane and inserting the actual or galvano-cautery in different directions, and then the skin flap replaced. A slough will very likely come away if it has been thoroughly done, but the result is sometimes most satisfactory, and the subsequent scarring much less than would have been expected. Erichsen in his work on surgery mentions a case he treated by using potassa cum calce in this way, where the nævus involved the whole thickness of the upper lip, but he appears to have done it through the skin or mucous membrane.

Crushing a nævus is a plan that has not much to recommend it. I practised it a short time ago on a child who had a deep nævus involving the whole thickness of the greater part of the upper lip. We turned back a flap of skin on the outside, and one of mucous membrane on the inside, after making an incision along the free border of the lip; having thus exposed the nævus it was crushed vigorously with artery forceps, in the hope that we might destroy it thus without hopelessly injuring the muscles of the lip. But the result was not satisfactory. The wound healed up immediately, but the nævus eventually did not seem much the worse, or rather the better for its treatment, and I have since operated on it with the actual cautery in the way I described before, this time with a much more satisfactory result.

Ligature was once a recognised method of dealing with deep nævi, especially those on the scalp, and in the older text-books on surgery rather elaborate directions are given as to the art of tying the ligatures subcutaneously. There are,

Digitized by Google

however, so many obvious objections to the method that I hardly think we need discuss it. I should mention, however, that in the case of a very large superficial nævus of the chest wall, where during excision the hæmorrhage was likely to be serious, I found a temporary ligature of considerable service. The ligatures were passed in the orthodox fashion under the affected area, but were inserted and brought out, not close to the growth, but a little distance from it on both sides, so that when they had been tied the incisions could be made through unaffected skin. These ligatures acted as a kind of tourniquet, and were cut one by one when the nævus had been removed and the hæmorrhage had been arrested with artery forceps as it occurred. For deep nævi, for which it was originally intended, ligatures might possibly be used in the same way, but the difficulty would always be to be sure they had all passed quite below the growth, otherwise they would be cut during its excision, and so their object frustrated.

Of the practice of *injecting* nævi with substances to cause the coagulation of the blood they contain, I know nothing. I have never done it or seen it done; various substances have been used for this purpose, such as perchloride of iron, carbolic acid, etc. It is possible it might be done successfully in a part where it would be feasible to put a clamp or temporary ligature round the growth to prevent the clotting spreading too far, or the clot separating, as in the lips or tongue. The danger of subsequent sloughing must, however, be great, and there are other methods of treatment more scientific and of more proved value.

It is said that small nævi may be cured by the repeated application of ethyl chloride or collodion, which act by causing contraction of the growth in much the same way as elastic pressure. It would be a tedious way of dealing with them at the best, and it is not much to be wondered at that the method is not much employed.

Neither these substances, nor those which ordinarily cause muscular contraction of the walls of the blood-vessels can have much effect on a nævus in this way, on account of the very small amount of muscular tissue which exists in them.

Of the treatment by *elastic pressure* it can only be said that if it be sufficient to cause effect, that effect would probably be a slough, and if the pressure were not sufficient to produce this, the nævus would probably be uneffected. To attempt to regulate the pressure of a bandage nicely enough to strike the mean between these, and so cure the nævus by keeping it empty of blood, is an undertaking that would hardly commend itself.

In conclusion, I must refer briefly to a method of treatment which probably exists more as a popular delusion than as a surgical proceeding, that by vaccination, and one is often asked by a mother whether this cannot be done, even when the growth is of some size. Owing to the thinness of the walls of the blood-vessels vaccinating on a nævus would be almost sure to cause a little bleeding, which would very likely prevent its success as far as vaccination was concerned, and even if it takes it can only cure the nævus by causing inflammation and sloughing. This might be only partially successful, and might even cause the rest of the growth to spread or multiply, and, I think, you will agree with me that it is much wiser to treat the nævus by excision or cauterising, and to perform the vaccination on some unaffected part of the body.

THE CLINICAL AND PATHOLOGICAL RELATIONS OF THE CHRONIC RHEUMATIC AND RHEUMATOID AFFECTIONS TO ACUTE INFECTIVE RHEUMATISM.

By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Physician and Lecturer on Medicine at St. Bartholomew's Hospital.

(*Concluded from p.* 5.)

We are now in face of the problem which particularly concerns us to-night, to wit, the dependence of rheumatoid arthritis proper on a toxæmia due to invasion by the specific cocci of true rheumatism, and have to express an opinion whether the varieties and degrees of the former, monarthritis, polyarthritis, dry arthritis, or arthritis with effusion, are the outcome of a toxæmia due to specific microbic invasion. Some evidence has been afforded by Dr. Bannatyne and others in support of this contention, and has already led to measures of treatment conceived to be appro-

Digitized by Google

priate for the destruction of such noxious influences.

It is certain that at the present time pathology in general is dominated by conceptions relating to parasitic invasion and the behaviour of various toxins thus generated, and it would be strange if a disorder such as rheumatoid arthritis were not now to be reconsidered from this point of view. It is only fair to add that these ideas are supported by some facts which have a close bearing on its pathogeny. The analogy of pyæmic and gonorrhœal arthritis must be considered, and especially minor degrees of toxæmic arthritis dependent on alveolar pyorrhœa, dental caries, influenzal, pneumococcal, and other toxins. The influence of disorders of the pelvic viscera in the female has been largely noted by observers, and by Dr. Ord especially, and uterine diseases, *post-partum* disorders, menorrhagia, and retained menstrual shreds and fluid have been often set down as antecedent if not provoking conditions of the rheumatoid state. The late Dr. Todd was much impressed with the view that these pelvic disorders let loose into the blood some peccant material which induced arthritis. On this hypothesis we might regard many cases of the disease as mild, or modified, but chronic forms of pyæmic arthritis, and, in the language of to-day, pronounce them as of infective or toxic origin. Accepting this view, we might fairly expect to find bacterial evidence in proof of it. And, certainly, on the principle of *sublata causa, tollitur effectus*, a removal of these existing causes, when possible, is found to act beneficially, provided the arthritis has not made too great progress.

Dr. Poynton has suggested to me as a "fascinating view" the idea that some cases of the disorder are of the nature of a malignant arthritis comparable to malignant rheumatic endocarditis. In a recent case of infective endocarditis under my care, the patient had signs of subacute arthritis in several joints, which passed off before death; but at the autopsy nothing was found amiss in the articulations. In another one now under my care, and in which pure cultures of streptococci were obtained from the blood, there have been mild forms of arthritis which have also subsided and left no further indications. If we regard infective endocarditis as a form of arterial pyæmia (Wilks), there is nothing remarkable in such an occurrence. These patients, at all events, almost invariably die within two or three months, and there is therefore no time for any chronic form of arthritis to be established. I have, however, known a case to last for twelve months without any sign of arthritis supervening. The articulations in many cases of toxæmia probably escape, because they are not specifically vulnerable; the subjects are not of the arthritic habit, or the joints have never previously been weakened by injury or disease. It has long been an axiom in surgery that any joint which fails to recover promptly from injury is declaratory of some habit of body or taint which renders it vulnerable, and the peculiar behaviour or lingering element of the inflammatory process not seldom indicates the precise nature of the constitutional state, possibly not previously suspected.

I do not think that up to the present time a case has been made out for a constant and specific toxic element, bacillary or otherwise, in the pathogeny of all cases of rheumatoid arthritis, and such specific element, if such there be, is therefore yet to be sought. If this be the case, it follows that we can trace no constant relation between this malady and true infective rheumatism. I say no *constant* or *definite* relation, for I have already declared for a basic relationship between disorders such as gout, rheumatism, and some other provocatives, as conditions to which persons of the arthritic diathesis are specifically prone. The most experienced pathologists allow that gout, frequently repeated rheumatism, gonorrhœal arthritis, and traumatic synovitis, with some other distinct morbid conditions, are capable of producing changes in joints which are indistinguishable *post mortem* from primary or so-called idiopathic rheumatoid arthritis. There is no going back upon such declarations, and we must accept the facts as stated, and I venture to repeat and reinforce them. We find difficulty, therefore, in propounding a pathogenic doctrine which would lead us to affirm that there is any single etiological unit which explains or comprehends the varied phenomena presented by the malady; but I am not prepared to add that this statement is likely to be the last pronouncement on the subject. We may fairly expect that more precise knowledge awaits us and those who will follow us, and it may hereafter be possible to differentiate the several varieties of the

Digitized by Google

disorders, and to indicate their precise dependence on the specific elements which respectively induce them.

I have hitherto said little respecting the theory which relegates this disorder to morbid conditions of the nervous system. This view followed the recognition of arthritic changes which resulted from injuries to the spinal cord, so-called neurarthritis, and the subject received renewed attention on the discovery of that strange variety of arthritis now known as Charcot's disease. Here was another form of joint-disorder arising in the course of spinal tabes, having peculiarities in respect of rapid and riotous disorganisation with much effusion, but yet presenting *post mortem* many of the features of rheumatoid arthritis, with an incapacity for reparative changes. The clinical concomitants of cases presenting this symptom appear to point very clearly to damage, involving a definite part of the medulla oblongata, and these led Dr. Buzzard to suggest that the sclerosing lesions of tabes had probably involved this part of the nervous system, a recognised centre of centres as we may regard it, presiding over cardiac and respiratory functions. And in this connection we have to regard the circulatory and articular elements or tissues together as motorial in their functions, and thus it has been assumed that there is a medullary centre presiding over the nutrition of joints, one which, under afferent impulses provoking irritation, may lead to trophic changes of a degenerative character in these structures. There are not wanting clinical facts which support such a hypothesis, amongst which may be mentioned the involvement of symmetrical joints in succession in some cases of rheumatoid arthritis, and the simultaneous occurrence of cardiac and articular disturbances in structures primarily motorial. We have, therefore, to take note of neurarthritic conditions in relation to the pathogeny, and the occasional association therewith of cardiac disorders, most commonly of arrhythmia or tachycardia. This theory of a medullary trophic neurosis is invoked by myself as part of the pathogeny of gout, whereby the specific determination to the joints, with degenerative changes, is constantly directed, but it is only fair to state that it is not yet generally accepted.*

In the endeavour to place our present amount of knowledge on the whole subject on a sound basis, it may be wisest to review it from a fresh standpoint, and to disburden ourselves of the ideas which have so long encumbered the inquiry relating to rheumatic or gouty elements as exclusive factors in the disease, to regard the plain facts of each example of it, and to start with the certain factor of arthritis, pure and simple, as a genus. The next step should be to find the exciting cause in each individual, to determine, that is, the species of arthritis. Careful inquiry into family and personal history should follow, and a study be made of all the concomitants and peculiarities of the disorder. Were this to be carried out, I believe that we should in many instances be able safely to relegate the manifestations to their primary cause. We should expect to find gouty and rheumatic antecedents and elements respectively, to discover strumous taint as sometimes modifying the phenomena, to determine that certain toxic matters were the specific provocatives in other cases, arising from possibly wide and diffused areas, or from a very restricted area somewhere in the body, or again from certain infective maladies through which the patient had passed, or was actually the subject of. It is right to state that we have as yet no knowledge as to the possible latency or dormant conditions of the germs which provoke true infective rheumatism. These may conceivably, as in the case of enteric fever and influenza, or of some other disorders, such as syphilis and the plasmodia of malaria, lie dormant, ready to be aroused by fresh provoking conditions. And these, with other intrusive germs, may by means of mixed and aggravated infection, lead to new developments in joints already susceptible and lowered in vitality. We await further researches on this point, and no less renewed examinations in regard to particulate invading elements in cases of active arthritis with effusion.

To follow out such a method as I suggest would at once entail our dropping the term "rheumatoid" as applicable generally to the disease, while we signalised the particular variety of provocative in accordance with the nature of the specific irritant in each instance. Although it is hard to part with old friends, yet to recast our views on these lines

* 'Brain,' April, 1888; and "Gout considered as a Tropho-Neurosis," 'Brit. Med. Journ.,' March 26th, 1881; "Pathogeny of Gout," address delivered at the XIIIth Internat. Med. Congress, 1900, in Paris.

Digitized by Google

would in no degree, as I conceive, disturb the usual conceptions which I lay stress upon respecting the diathetic tendency and proclivity of individuals liable to chronic forms of arthritis, since this habit of body is not merely and exclusively vulnerable to gouty or rheumatic conditions. It reacts badly to other provocatives, while it is largely protective against tubercular invasion or developments. We have to regard the onset of arthritis under many circumstances as the alighting of disease on areas of tissue that are already inherently vulnerable, or which have become so by injury, strain, overuse, or antecedent disorder. We now know that antecedent maladies weaken one or more organs, and this predisposes them to infective diseases. Influenza and scarlet fever may be noted as instances leading to such disorders. Pyogenic microbes certainly reinforce the actions of others in the body, and thus we have often to deal with mixed infection, whose ravages exceed those of any single toxin. In this way the action of cold or heat, or of changes of atmospheric environment, predispose to the action of definite infections.

At the present time, then, it appears to be unjustifiable to declare for any unity of pathogeny in respect of the condition we term rheumatoid arthritis, or to regard all examples of the disease as depending on any uniform or definite cause.

In the treatment of most examples of it, our therapeutical indications appear to be to restore the general level of health of the patient, and to place him in such an environment as may best secure this. Special indications will be suggested by the features of the case which declare the origin and provocative of it, and we are not warranted in assuming that any particular method of diet, or treatment at any particular Spa, is required for all cases presenting the bare symptoms of chronic arthritis. Here, as in all cases, we have to treat patients, and not diseases.

I have ventured to draw up a scheme showing how the disorder may be induced. It is wide enough, and in parts hypothetical, but it may be suggestive and helpful.

In any case our duty is to try and be as exact as we can, and to endeavour to study carefully the precise features of each one as it comes before us, to put aside old prejudices, and to keep an open mind.

CHRONIC ARTHRITIS.

Basic arthritic diathesis or habit of body, with instability of medullary centres which preside over and control the locomotory apparatus, articular and circulatory (? transmissible central neurosis).

The body specifically vulnerable to certain influences, infections, toxins, trauma, strain.

Tendency of these to be determined to the joints.

Excitants and provocatives of arthritis in lingering or disabling forms:

- (*a*) Infective (acute) rheumatism.
- (*b*) Auto-toxin of gout (urichæmia of all grades in the gouty.)
- (*c*) Gonococcal toxin.
- (*d*) Pneumococcal toxin.
- (*e*) Influenzal toxin.
- (*f*) Pyæmia (of all grades of intensity), *e. g.* pelvic, alveolar, carious teeth, infective endocarditis, etc.
- (*g*) Syphilitic toxin.
- (*h*) Hæmophilia (gouty relationship).
- (*i*) Mixed infection.
  - Scarlatinal and rheumatic.
  - Scarlatinal and pyæmic.
  - Tubercular and rheumatic.
  - Tubercular and gouty.
  - Tubercular and syphilitic.
  - Rheumatic and gouty.
- (*j*) Malaria.
- (*k*) Dysentery.
- (*l*) Morbilli?
- (*m*) Senile influences.

Neurarthritis;

- (*α*) Primary.
- (*β*) Secondary (reflex).
- (*γ*) Charcot's disease.

Otitis and Cerebro-spinal Meningitis ('Bull. Soc. Hôpitaux de Paris').—Besides the meningitis that develops locally from direct communication with a focus in the ear, Vaquez and Lermoyez have observed cases, and three are described in detail, in which an old chronic otitis seemed to have been the origin of a generalised cerebro-spinal meningitis—a remote infection in which the spinal predominated over the cerebral symptoms.—*Journ. Amer. Med. Assoc.*, April 13th.

Digitized by Google

A CLINICAL LECTURE.

By H. W. SYERS, M.A., M.D.Cantab.,
Physician to Out-Patients, Great Northern Central Hospital.

GENTLEMEN,—In everyday practice it is the thorough appreciation of the most numerous forms of disease, which is above all things essential. It is no doubt very desirable to have a knowledge of the physical signs and symptoms of a recondite malady, such as is by any possibility hardly ever met with in practical work, and when this special knowledge is combined with the power of making accurate diagnosis in every event, all things are, of course, as they should be in the best of all possible worlds. In out-patient practice, however, the circumstances are such that it is not always possible to make very accurate diagnoses. Pressure of time is great in this work; the number of cases to be seen is very large, and in a preponderating majority physical signs are conspicuous by their absence.

This latter fact leads me to dwell a moment upon some of the disadvantages and some of the advantages of this kind of practice. Amongst the former is, of course, the fact that an immense proportion of these cases are trivial in the extreme; they are not in the real sense of the term in need of medical treatment. What they require is better food, better hygienic surroundings, less anxiety.

The *res angusta domi* is the real cause of the morbid feelings, pains, and aches of which they complain, and it is absurd to suppose that the administration of the "three times a day" mixture can have any, even the slightest, effect in amending their condition. Another great disadvantage is the fleeting and irregular nature of the attendance of out-patients. A case of particular interest or importance will turn up once and be no more seen, whereas those whose attendance is totally unnecessary will come with the most wearisome pertinacity.

I need scarcely say that any deductions as to the action of drugs which concern therapeutics in general are in the highest degree untrustworthy. To determine the real action of a remedy requires the closest attention and the comparison of cases; both of these conditions are entirely unattainable in the out-patient department. The utmost that can be hoped for is to obtain the sort of information, upon which, however, much of therapeutics is based, which is really no evidence at all of the facts it is too often held to prove. The patient takes the medicine, and next time he is ready with the information that he felt "very much better" for having taken it. By a certain order of mind, which, by the way, seems to be particularly common amongst those who devote themselves to investigating the healing properties of drugs, this statement would be regarded as of the highest importance, and as justifying the inference that such and such a drug was of great service in the treatment of such and such a disease. This hopeless confusion of the *post* and *propter* is the bane of therapeutics, and no doubt to a very large extent accounts for the incessant outpouring of new drugs, each of which has its little day, and is thereafter consigned to oblivion.

Such, then, are some of the drawbacks to out-patient practice. Now let us for a moment consider some of its advantages. To the observer well skilled in physical diagnosis these are not small; he learns to recognise the physiognomy of disease, and this is a most important matter. By the way the patient opens the door, by the way he states his case, by the way he leaves the room—in all these ways will the experienced observer be guided in his diagnosis. The very tone of the voice is helpful, and there is scarcely a gesture that has not its meaning. I need scarcely say how important it is for everyone to do his utmost to acquire the faculty of physiognomical diagnosis, and certainly I know of no place where this faculty can be attained more quickly and more accurately than in the out-patient room of a large London hospital.

The second point in favour of this kind of practice is that such great variety exists in the different forms of disease, such variety, indeed, that without a certain effort a tendency to discursiveness is apt to arise. From a heart case the attention is called to an abdominal disease, and thence perhaps the next two cases may lead the observer on direct to the larynx, and to the skin. But in this way the faculties are kept keen, and the necessity of taking a *comprehensive* view of each case is firmly fixed in the mind. Lastly, that very preponderance of trivial cases leads the out-patient practitioner, on the one hand, not to dismiss

Digitized by Google

as frivolous every complaint unattended with physical signs, and on the other prevents him from wasting time over examinations which are totally unnecessary. Further, by the constant study of such cases he may ascertain in particular instances upon what faulty general condition the special complaint may depend—a knowledge difficult to acquire, but which may be of the greatest value in practice.

I now proceed to point out some errors of diagnosis which are very easily made, and which it is useful to be acquainted with on account of their frequent occurrence.

First of all, I have no doubt that it has happened to you, as it has frequently to myself, to mistake at the first glance regurgitant aortic disease for chronic phthisis. The resemblance between these maladies is really astonishing, and I doubt very much if it is possible by physiognomical diagnosis alone to differentiate them. In both cases the face is thin, the skin pallid, the expression slightly drawn and anxious Over and over again I have mentally diagnosed a case as one of phthisis, and on proceeding to examination have found that I was entirely mistaken. On the doctrine of chances, of course, any given case is far more likely to be one of phthisis than of aortic reflux, as the former malady is far more common than the latter. I may add that this resemblance between the two affections has been noticed by several observers, but I have not found that the fact of the physiognomy of phthisis and of incompetence of the aortic valves being identical, is at all well recognised.

Another physiognomical resemblance is between myxœdema and Bright's disease, and there are cases which at the first glance it is impossible to define as between these maladies. I have repeatedly been unable to settle the question until I had made a thorough examination, including that of the urine.

Once more, it must have happened that you have seen patients suffering from dyspnœa, with cyanosed face, and much acceleration of the respiration, and movement of the alæ nasi, in whom it was absolutely impossible to say that the case was not one of failing, or failed compensation. Yet an examination of the patient proved that the heart was perfectly sound, and that the case was no other than acute bronchitis.

It is very difficult, indeed, impossible, in some cases, to know whether a patient is the victim of hysteria, or of serious cerebral disease. And in the out-patient room it is utterly out of the question to make the diagnosis as between the two affections, and yet what an error is made if what is incurable brain disease should be diagnosed as hysteria! I recall a case in which nearly every possible gross symptom of hysteria was present, and yet the patient was suffering from a large tumour of the frontal lobe. I should add that this was not an out-patient case, but the history is none the less instructive.

The resemblance superficially between general paralysis of the insane, when of some development, and drunkenness, must have struck all of you. I have myself been implored by a lady to dismiss a tradesman who had called upon her with reference to some business—the man being, to use her expression, "quite tipsy." A moment's consideration showed me that the poor fellow was perfectly sober, the thickness of speech and tremor of the lips being wholly the result of the disease.

Errors in diagnosis of renal disease are easily fallen into in the out-patient room. It should never be forgotten that the symptoms of chronic disease of the kidneys are often misleading, and would tend to show that it is a malady of some other organ which is present. Patients suffering from granular kidney often complain of severe headache, or troublesome diarrhœa, or merely of loss of appetite. In all such cases it should be a cardinal rule to investigate the arterial system generally, and the urine in particular. It is perfectly possible with the stethoscope alone to diagnose chronic renal disease, for the changes in the heart-sounds which accompany those in the kidney are absolutely characteristic.

Whenever, therefore, the first sound at the apex is reduplicated—the two ventricles not acting synchronously—or if there is an accentuation of the aortic sound, or a reduplication of the whole second sound, attention should at once be directed to the radial artery and the kidney. In nine cases out of ten the former will be found of high tension, or, what is the same thing, the pulse will be incompressible; and the secretion of the latter will contain a trace of albumen.

I only incidentally mention eczema and itching of the external genital organs, most often com-

Digitized by Google

plained of in women. It is, of course, absolutely necessary to at once examine the urine for sugar, in order to avoid the danger of a disastrous mistake. The same examination should be performed if a patient tells us, as is sometimes the case, that he always finds flies hovering over and settled in the chamber utensil when the latter has been used.

Patients not infrequently come to the out-patient room with the face covered with a red rash which may, more or less, obliterate their features. Be very careful not to say that the cutaneous affection is erysipelas, to which there is considerable superficial resemblance.

These cases are nearly always *acute eczema*, but nothing but a careful examination and a search for the initial lesion will settle the question of the real nature of the rash. The essential lesion of eczema is a vesicle, and you will find that the characteristic minute vesicle will invariably be forthcoming when a thorough search is made. The tendency to confuse these complaints is extremely common, and their great resemblance accounts for this.

Another mistake not seldom made is the confusion of syphilitic scaly rashes with psoriasis; and yet there is the greatest possible difference between the two lesions. I am inclined to think that much or most of this confusion is due to the habit of speaking of "syphilitic psoriasis," which is not uncommon at the present time. If the term "scaly syphilide" were alone used for the specific affection, all such mistakes would cease. Psoriasis is one disease, syphilis another and totally different one; to join the two distinct maladies in one term is to court and increase classificatory chaos. This tendency to confound different cutaneous maladies, and to unite those which are quite distinct has always been the bane of cutaneous medicine. The rash of psoriasis is fresh-coloured, is very scaly, is most marked on the extensor surface, does not leave much pigmentation, except on the lower extremities in old-standing cases, and is frequently inclined to itch. In all these points it is contrasted with the scaly syphilide. When it is further remembered that the places of election of psoriasis are the point of the elbow and the front of the knee, also the scalp, it is obvious that it only requires a thorough examination and a little patience to make a sound diagnosis.

A patient comes to the out-patient room cyanosed, with rapid breathing and the complaint of shortness of breath and cough. All this dates from a few weeks only. On physical examination nothing is discovered beyond the signs of acute general bronchitis. No dulness is anywhere detectable, and very frequently rhonchus and sibilus are alone audible. I have seen cases such as this typical instance, diagnosed again and again as acute general bronchitis. This is of course a correct diagnosis, but it is a totally insufficient one. The disease is acute tuberculosis, and I need scarcely point out the serious mistake which is made when the diagnosis goes no further than the auscultatory signs. But how is it possible to establish the fact of the serious disease, tuberculosis, being present? By the prominence of cyanosis. The lips are remarkably blue in these cases, and this blueness together with the rapidity of the breathing are two most important points, which are only too often entirely overlooked. You will, of course, remember, that I said at the outset that the symptoms were of a few weeks duration only. I need not emphasise the importance of this remark, suffice it to say that emphysema and chronic bronchitis are both excluded.

Of the large number of cases seen in this room a considerable proportion consist of anæmic young women. So constant is the stream of these patients that one is rather apt to see such a case, to make merely a superficial examination, usually limited to the detection of cardiac murmurs, and to dismiss the patient with iron and aperients. Yet it should never be forgotten that, in *young* women especially, anæmia and amenorrhœa may be the very earliest indications of phthisis. So that in all such cases it is as well to inquire particularly if the patient has any cough: if the reply is in the affirmative a minute and careful examination of the pulmonary apices should immediately follow.

When a patient states that he or she is, and has been for some months, suffering from troublesome diarrhœa or "dysentery," never neglect to make a digital examination of the rectum. If you fail to do this you will fall into the error of passing over malignant disease of the rectum, for I have invariably found that symptoms such as these are due to cancer of the rectum. Further, you must not suppose that malignant disease is excluded on account of the youth of the patient. I have

Digitized by Google

repeatedly met with cancer of the rectum in persons between twenty and thirty, and it may certainly occur even at an earlier age.

Empyema in children is a malady frequently overlooked or wrongly diagnosed A child, wasted and anæmic, is often brought to this room with the history that it has been languid and dull, with failing appetite, but without cough or any particular chest symptom. Under these circumstances attention is more likely to be directed to the abdomen than to the chest, and a completely erroneous diagnosis may be made. But even if the chest is carefully examined the case is often misinterpreted. It is not sufficiently recognised that the signs of empyema in children are very different from those of the same affection in adults. In children you must not expect to obtain the marked and definite dulness noticed in adult life, neither must you rely on the auscultatory signs. These are very often those of consolidation rather than of pus, for it is by no means unusual to hear bronchial breathing over children's empyemata. Very light percussion with one finger will often elicit a higher pitched note over the affected area, and this with some alteration in the breath-sound will be sufficient to justify the use of the exploring syringe. I need scarcely say that no importance whatever attaches to the temperature, in the diagnosis of these cases; for it is often raised when the fluid is clear, and is just as often normal when pus is present. With practice it is possible to recognise cases of empyema in children by the naked eye. They have a more or less characteristic yellow-looking skin, are lethargic and dull frequently, but by no means always, and the terminal phalanges on the affected side are apt to be clubbed if the disease is of any duration.

Not long ago a young man presented himself complaining of the sudden appearance of a "red rash" over his chest, stomach, and legs. On examination it was found that his statement was quite accurate—there was such a rash in the situation indicated. The eruption resembled very strongly indeed that of scarlatina, and the temperature was taken and his throat examined. The temperature was normal, as also was the condition of the throat. This being so the next inquiry was concerning the presence of venereal disease, and at once the nature of this rash became manifest. He had gonorrhœa, and had been taking sandal-wood oil for it. It is, I believe, somewhat unusual for a scarlatiniform rash to follow the administration of sandal-wood oil, though it is not uncommon after swallowing copaiba balsam. This case is one of the large class of drug rashes, and apropos of them I ought also to mention those eruptions which are factitious. Whenever you meet with a rash which you cannot classify, which differs from any skin disease with which you are acquainted, and which comes and goes in an unaccountable manner, always suspect a factitious rash, the more so if the patient is a young female. There is no limit to the lengths to which some young hysterical women will go in these cases, and the most curious and anomalous eruptions or skin lesions may result. Herein consists the difference between a drug rash and a factitious one. The former bears more or less likeness to some definite lesion, the latter is a thing which is altogether aberrant.

We are taught by the experience of out-patient practice to be very chary of the diagnosis of "obstruction of the bowels." In nine cases out of ten in which we are informed by patients that they are suffering from this serious complaint, investigation will prove that they are constipated. The serious form of obstruction of the bowels will almost invariably, in my experience, resolve itself into acute general peritonitis, in the majority of cases caused by perforation of the vermiform appendix by a fæcal calculus, at all events in young males.

The pernicious and illogical use of the word "influenza" has led to a distressing habit, on the part of out-patients, of constantly employing this most objectionable term. They say that they have suffered or are suffering from an attack of this ubiquitous malady, and it is often impossible to disabuse their minds of the fact that they are totally mistaken. Doubtless the manner in which the influenza epidemic has been exploited in the press and otherwise, has led to the incessant cropping up of the word in the out-patient room. In my experience the term "influenza" is used —(1) by hypochondriacs whose attention has been riveted on the discussion of the affection in the daily press; (2) by those suffering from some real and definite disease, under a complete misapprehension; (3) as a means of escaping duties, in others words by malingerers. Of true, real and

Digitized by Google

substantive "influenza" you will have seen little or nothing in this room. This is to me a distasteful topic, but the parrot-cry of "influenza," so often heard amongst out-patients, renders a reference to this objectionable matter necessary.

I have now exceeded the limits I had originally intended to confine myself within, and yet the subject is by no means exhausted. I hope to resume it at some future time.

SPRING FINGER.

The Doigt à Ressort of the French, and the schnellender Finger of the Germans.

The record of a case in which the affected part was exposed by operation, a contracted sheath stretched, and the patient cured.

By E. NOBLE SMITH, F.R.C.S.Edin., L.R.C.P.Lond.,
Senior Surgeon to the City Orthopædic Hospital, London.

A VERY good description of this affection was written by Mr. Henry Reeves in 1885.* Previously few English writers had described this troublesome condition, but several Continental surgeons had referred to the subject.

"Spring finger" consists in a partial obstruction to free flexion or extension (or both) of a thumb or finger, occurring at one particular joint, the difficulty being presently overcome, and the flexion or extension completed with a snap.

The obstruction generally occurs at a meta-carpo-phalangeal articulation, and is sometimes so persistent that the patient is induced to help the completion of flexion or extension by pressing upon the digit with his other hand.

Causes.—In the majority of the recorded cases, rheumatism is said to have been present, in others gout. The affection has also been attributed to injury, such as hyper-extension.

According to the statistics of 33 affected digits, the ring finger suffered 11 times, the thumb 11 times, the middle finger 9 times, the little finger once, and the index finger once. These cases were divided among fifteen females and five males, some having two or more digits involved; these include Mr. Anderson's records.

In two cases, a male of forty-nine and a female of sixty-eight, the affection was preceded by some degree of Dupuytren's contraction of the palmar fascia.* In the others there was no history of this affection.

Hoffa† refers to 121 cases recorded in literature and collected by Necker, which include 150 affected fingers.

Of 113 patients, of whom the sex was given, 62 were females and 51 males, so that the affection seems to be pretty equally distributed as regards sex.

The age was found to vary greatly. In two cases the affection was congenital, and Hoffa observed a case in a child aged six months. The majority of the cases occurred in middle life, but some in old age.

The middle finger was mostly affected, next the ring finger, the thumb, the little finger, and least often the forefinger.

In several cases the affection was symmetrical in the two hands, the two ring fingers being mostly affected. The right hand was more often involved than the left.

Pathological condition.—The fact that the obstruction occurs at one exact point, both in flexion and extension, indicates the existence of a fixed lesion, and contra-indicates the presence of a loose or pendulous body in the joint. In many of the recorded cases a nodule has been felt, apparently a thickening of the tendon at one point. Nelaton, Notta,‡ Menzel,§ and Hyrtl, all held the view that such thickening existed, and caused the obstruction which met with resistance in passing through the tendinous sheath, and the last-named author and also Berger thought that this sheath might be narrowed. Dr. Carlier, who has written a book upon the subject, records two cases in which a node could be apparently detected by the finger of the surgeon, but upon dissection no thickening of the tendon was found, but "Leisering, of Hamburg,

* H. Reeves, 'Bodily Deformities' (Lewis, London, 1885).

* Bernhard, "Beitrag zur Lehre von schnellender Finger," 'Centralblatt für Neukrank.,' No. 5, 1884.

† Hoffa, 'Lehrbuch der orthopädischen Chirurgie,' Stuttgart, 1898.

‡ Notta, 'Archives générales de médecine,' 1850, series iv, tom. xxiv, p. 142, etc.

§ Menzel, "Ueber schnellender Finger," 'Centralblatt für Chirurgie,' 1874, No. 22.

Digitized by Google

actually exposed a nodosity in the profundis tendon at the level of the point at which it entered the canal of the flexor sublimis, excised it, and cured the disease." In another case a fringe-like tumour was discovered "springing from the synovial covering of the flexor sublimis" (Anderson*).

An alteration in the shape of the articular surface of the joint has also been suggested as a cause, and this seems to be the case in a similar condition occurring in the mid-phalangeal joint of the great toe in a young lady, a patient of mine, who was also suffering from contraction of the plantar fascia. I relieved the contracted fascia by subcutaneous section, but I do not expect this to do any good to the "spring toe." This condition in the toes has been noted before, especially in association with hammer toe, and is supposed to be due to a transverse ridge upon the surface of the proximal phalanx.

Osseous excresences, the result or not of rheumatoid arthritis, and spasm of muscles have also been referred to by Carlier as causes.

Roser and Lisfranc have suggested that some change in the tendon, involving a roughening or thickening, is the cause of the affection.

Reeves thinks that a thickening of the tendinous sheaths may be the cause, and he points out that in the case of the thumb the affection "seems due to a pari-articular inflammation of the tendinous sheaths, and especially at the region of the metacarpo-phalangeal joint. The groove in which the flexor longus pollicis runs is," he remarks, "at this spot limited by the sesamoid bones, and bridged over by a firm fibrous structure, converting it into a canal, and it seems, anatomically, highly probable that the slightest thickening of the tendon in its synovial sheath would at this spot lead to obstruction in its motions."

In the other digits, in which similar firm osteo-fibrous canals are strengthened by the transverse and crucial bands, he suggests that it is probable that the obstruction may be due either to thickening of the tendons or to narrowing of the canal alone.

Hoffa refers to the researches of Schoenborn as showing a variety of causes, and states that Bruns, Leisrink, Weisinger, Carlier, and Lick found actual circumscribed thickening of the tendon.

Steinthal found contraction of the lateral ligaments.

In Schoenborn's case a strand of connective tissue crossed the tendons of the two flexors, and division of this strand gave relief.

It seems probable that each of these different causes may exist in particular cases, but in the instance of this affection which I am about to record the obstruction seems to have been due alone to constriction of the sheath.

Miss S—, æt. 18, was brought to me on October 4th, 1899. She had suffered from spring finger from the previous January. At that time she had been practising with a mandoline for three months, and suddenly, while dressing her hair, the ring finger of her left hand became affected at the metacarpo-phalangeal joint, and had so continued ever since whenever she flexed or extended the finger. Her mother had been troubled with the same condition in a slight degree for many years, the middle finger of the right hand being affected.

I could feel nothing abnormal in the tendon or its neighbourhood, and I proposed an operation. Upon cutting down upon the tendon, no enlargement could be detected at any point, but upon passing a probe beneath the tendon sheath, the passage was found to be very restricted, so that it was with difficulty that the probe could be made to pass. I inserted a blunt hook within the constricted sheath, and stretched it forcibly.

The wound healed by first intention, and the affection was perfectly cured. Once only, about three weeks later, was there a slight jerk in flexion, but since then the movement of the digit has remained perfectly normal.

The mother of this patient wrote to me on November 4th, 1900 (that is thirteen months after the operation):—"I am pleased to tell you my daughter's finger is a perfect cure, except that it is a little stiff."

I again wrote to the mother in January, 1901, asking to what degree the stiffness existed, and received the following reply:—". . . It is only stiff in the early morning, and when this goes off, after an hour or so, she can use it the same as her other fingers."

I think we may fairly expect this slight stiffness to disappear in time.

* Anderson, 'Deformities of Fingers and Toes,' Churchill, 1897.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 445. WEDNESDAY, MAY 8, 1901. Vol. XVIII. No. 3.

CONTENTS.

PAGE

*A Clinical Lecture on Cerebral Diplegia in Children. By F. E. BATTEN, M.A., M.D., M.R.C.P. 33

*The Clinical and Pathological Relations of the Chronic Rheumatic and Rheumatoid Affections to Acute Infective Rheumatism. ARCHIBALD E. GARROD, M.A., M.D., F.R.C.P. 38

*The Infectivity of Acute Rheumatism, with Especial Reference to the Chronic Types of the Disease. By F. J. POYNTON, M.D., M.R.C.P., and ALEXANDER PAINE, M.D. 41

*The Toxic Origin of Rheumatoid Arthritis. By N. C. MACNAMARA, F.R.C.S. 44

*The Relationship of Acute Rheumatism to Chronic Rheumatoid Arthritis. By C. O. HAWTHORNE, M.D., M.R.C.P. 46

Specially reported for the Clinical Journal. Revised by the Author.

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

CEREBRAL DIPLEGIA IN CHILDREN.

Delivered at the Hospital for Sick Children, Great Ormond Street, February 21st, 1901,

By F. E. BATTEN, M.A., M.D., M.R.C.P.,

Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen Square.

GENTLEMEN,—The use of the term cerebral diplegia has by some authors been limited to congenital bilateral lesion of the cerebral hemispheres. I shall, however, use the term in its widest sense to include a group of cases which clinically present similar symptoms — whether congenital or acquired—in which there is evidence of affection of both cerebral hemispheres. In the first place, then, I would put before you a clinical picture of the disease in its most typical form, and then proceed to the consideration of the various factors giving rise to this condition, to the various forms of the disease, and, finally, will touch upon the very interesting pathological condition which is found in these cases.

Symptomatology.—The first symptom may be noticed soon after birth, or more commonly at the time when the child should begin to walk; in other cases, however, nothing abnormal is detected till adolescence, or even till adult life is reached. Let us take, for example, a child of two years of age to whom our attention is first called because it is unable to walk. On examining a marked case of this sort it is found that there is general rigidity of the limbs. The legs are extended, the thighs adducted, the feet pointed. There is often some flexion at the hip-joint which causes the child when attempting to walk to bend the trunk forward. The muscles are hard and firm, and often well developed; so marked is this feature at times that there seems to be actually hypertrophy.

Passing next to the arms, we frequently find

Digitized by Google

them rigid, often in a flexed position, and strongly adducted towards the middle line. The hands are flexed at the wrists, and in extreme cases the fingers and thumbs are so tightly closed that they are driven into the palms; occasionally one sees the thumb inverted into the palm, and the fingers closed over it. The position of the body tends to vary somewhat; sometimes it is extension, sometimes flexion, but that which is most commonly seen is one in which there is flexion of the trunk, and in these cases the head is generally somewhat flexed on the thorax. It is in these cases also that the flexion of the thighs and knees is most commonly seen.

Passing to the face: we find the muscles of the face are also somewhat rigid, giving rise to a curious fixed expression, and it not uncommonly happens that when the facial muscles are affected nystagmus is also present. Other cases show no nystagmus. In cases of the severe forms here described, there is usually almost complete loss of power over the limbs. The child is quite unable to walk, and is practically unable to move its limbs, but in the less severe cases all movements are possible, though they are generally clumsy and slowly performed. In those cases where the child can walk the gait is that typical cross-legged gait which has so frequently been described in cerebral diplegia, and when once seen can never fail to be easily recognised. In some of these cases athetoid movements are present; in others, again, movements are seen which are very similar to chorea, and as a matter of fact many of these slighter cases are put down to chorea, and their distinction lies in their extreme chronicity, and in their having no association with rheumatism. With regard to the mental condition of these patients, it is usually somewhat defective, but on this point again there is considerable variation, and the children are frequently described by the mother as being extremely bright. On questioning, however, one finds that their education is often defective, and the mother attributes this to their being so intelligent that they have not been sent to school. As a rule we may take it that there is some mental defect, varying greatly in degree. In some children it is extremely marked, while on the other hand it is so slight as not to be noticed until the child gets to the time when it should go to school, and then the discovery is made that they cannot learn or do their work as well as the normal child. The speech in some children is absolutely unaffected, while in others they are not only late in learning to talk, but it is slow and scanning, and the power of expressing their ideas in words is very limited. Other cases show rather a defect in articulation, probably due to the bilateral affection of the hemispheres, which imparts a more or less nasal tone to the voice. In certain cases convulsions may be present, and they are usually generalised, but it is only in the severe cases that such convulsions are met with, and in the slighter cases they do not occur. In the eyes, again, one finds a variety of lesions; in the majority of cases the vision is good. In some cases primary optic atrophy occurs, and it may be so advanced as to render the child perfectly blind. One curious condition which I have seen associated with these cerebral diplegias is the occurrence of congenital cataract, which was noted in two members of one family, in whom symptoms of cerebral diplegia were present. In another form of the disease, in which the child is amblyopic, it is found on examining the fundus that at the macula there is a cherry-red spot in the centre of a pale zone. These cases form a distinct group, and are known under the title "infantile cerebral degeneration." So far as present evidence goes they occur only in children of Jewish parents. The condition is a progressive one, and was first described by Waren Tay. With regard to the other ocular symptoms, inequality of pupils is not uncommon, reaction to light is generally perfect, except when optic atrophy is present, and then the pupils do not react well. One finds nystagmus in those cases in which the condition has passed high up, and has affected the face. Strabismus is most common in this class of case. When asked to protrude the tongue they do it extremely slowly, and sometimes they are quite unable to accomplish it. The palate, as a rule, moves well on reflex stimulation, but not well on phonation. As you are well aware, in all bilateral lesions of the hemispheres one gets a certain number of bulbar symptoms, and it is most common with these children for the mothers to tell you that they have very great difficulty in feeding them. Some are extremely slow in taking their food, and will only take liquids, and no solids. This condition, as a rule, is more marked

Digitized by Google

the younger the child is, but it will persist so that the child will remain on milk diet till it is four years of age, and even then all its food will have to be softened before the child can swallow it. You will notice that this manifestation is the exact reverse of what occurs in other affections, such as paralysis of the palate after diphtheria, where solid food is more easily swallowed than liquid. The shape and size of the skull are generally normal; it may, however, be either hydrocephalic or microcephalic. The spine often shows some deformity, and there may be kyphosis, lordosis, or scoliosis. In some cases deformity and alteration in the shape of the feet is met with, the plantar arch is heightened, the big toe is extended and retracted, and a position of pes cavus is produced. Whenever this deformity is seen it should suggest the possibility of some affections of the pyramidal system, and it is due to altered tones of the muscles. In other cases there is a tendency for the feet to assume the deformity known as equino-varus. No defect in sensation is present.

Turning to the reflexes, we find that the knee-jerks are extremely exaggerated, and the ankle-clonus is often present; but if there is much rigidity of the legs it is not easy to obtain it. The patellar clonus is sometimes present, and the plantar reflex gives as a rule the typical extensor response. The arm-jerks are easily procured, and in cases where the face is affected, one finds a jaw clonus.

We now come to the ætiology of the disease; it is common to attribute the disease to injury received at birth, either by instruments used in delivery, prolonged labour, precipitated labour, or asphyxia. One cannot here enter into a discussion of the importance of these various factors; that they have their influence one cannot well doubt, but that they play that important *rôle* in the production of the disease which is usually ascribed to them may very well be questioned. But the difficulty of birth may be dependent upon two factors: first, there is the size of the child's head in relation to the pelvis; and secondly, the condition of the mother, and this latter point brings us to the next most important ætiological factor, viz. the maternal condition during pregnancy and labour. Parents themselves always attach the greatest importance to such factors, and the weight of evidence certainly tends to swing the balance in that direction. But of all the constitutional ailments which affect the parents, none play so important a part as syphilis. Here one has probably the direct effect of the toxin acting on the fœtus before birth. In many maternal ailments considerable weakness is produced, which tends to prolong labour, and this will probably bring the first ætiological factor, *i. e.* the difficulty of labour, into prominence. The third important factor in connection with the ætiology is its occurrence subsequent to some acute illness, either one of the acute specific fevers such as typhoid, diphtheria, or influenza, and the condition known as acute polio-encephalitis.

Having considered the general symptomatology and ætiology of the disease, one passes to the consideration of the various types. The first type is that known as the congenital spastic paraplegia dating from birth, and often associated with more or less mental impairment; and under this heading one includes also all cases of generalised rigidity dating from birth, whether associated with choreiform and athetoid movements or not. Next one has a progressive form of generalised rigidity coming on at a varying period after birth and tending to fatal issue, a rather rapid degeneration taking place in children between one and two years of age. Closely allied to the last form is the progressive form of degeneration seen in Jewish children, who become amblyopic and present certain characteristic changes at the macula to which I have already referred.

Then there is the family form, in which several members of the family are affected, the children being as a rule normal till about the eighth year of life, and then gradually becoming spastic; the disease is very slowly progressive. These cases belong to the same category as the spastic family paraplegia which is met with in adult life.

Then there is the form of the disease closely connected with congenital syphilis running a steadily progressive course with considerable mental impairment, and that condition is frequently described under the title of "juvenile general paralysis."

The last form is acquired, occurring after some acute illness, which gives rise to bilateral affection of the cortex; this form is not as a rule progressive, but tends rather to improve. I am perfectly aware that such a classification is an

Digitized by Google

artificial one, and that intermediate forms exist, but it is necessary to have some such classification when one makes the term diplegic so comprehensive, for among those diseases classed under diplegia one finds cases, some of which are rapidly progressive and fatal, others which are stationary or very slowly progressive, and others again which tend to improve.

Having thus given an outline of the general features of the disease, I will now endeavour to illustrate some of the points by the cases I have here this afternoon, and subsequently I will deal with the pathology and prognosis of the disease.

Case 1.—This child was brought to me some three years ago, when the child was two years of age, with a history that she had never been able to walk. She was quite intelligent, could talk well, and was clean in her habits. She was the third of four children, all the others being healthy. The labour had been protracted, and delivery was effected with forceps. On examination of this child you will notice that the legs are rigid; the child can support her weight, and when assisted can make some attempt at progression. The feet tend to assume the position of pes cavus, with a tendency to equinus, so that the child walks on tiptoe. There is no loss of sensation, and the sphincters act normally; the knee-jerks are greatly increased, and ankle-clonus is usually present, and the plantar reflex gives the extensor response described by Babinski, the big toe being drawn back. This case, then, is a typical example of the disease usually known under the title "congenital spastic paraplegia," or Little's disease, so called from the name of the person who first described it. There are, however, one or two other points to be noted in this case. You will remark that the child uses her right arm well, but when using the left arm the movements are clumsily performed, and, apart from the clumsiness on voluntary movement, there are frequent inco-ordinate choreiform movements. This condition shows a transition stage to that type of the disease known as "choreiform." This child has improved since she has been under observation, and more especially so since she has been able to get about in a go-cart.

Case 2.—The second case presents a somewhat different form of the disease. The child is aged two years, and is the seventh of eight children; all the others are in normal health. The birth was long and difficult, and the child was born at the seventh month; the mother was ill at the time; it was a breech presentation; no instruments were used. The child has never been able to walk, but progresses along the floor by alternately rotating the pelvis on the spine, and for this movement the child does not use its hands, and the legs are somewhat rigid, but are flexed instead of being extended. The knee-jerks are active and increased, and the plantar reflex gives the extensor response. There is not very marked rigidity of the legs in this case.

Case 3.—The next case is a boy four years of age, who has never been able to walk properly; he is the second of three children. Though born at the seventh month labour was quite natural. The marked squint has been present since the child was three months old, and is attributed to "cutting his teeth." The child is mentally dull, and not very clean in its habits, and walks in a manner typical of the disease. He has not that marked adduction of the thighs which you saw in the first case. His knees are somewhat bent, and he drags his toes. There is a certain amount of rigidity of the legs, very active knee-jerks, and the plantar reflex gives the extensor response, but there is no ankle-clonus. At his age he ought to have lost the extensor response, which is normally present in infants, and have the typical flexion of the big toe. The arms are unaffected. The skull is fairly well developed, but narrow in the frontal region.

Case 4.—The next case is a child three years of age, who has been under observation for a considerable time. At the age of one year the child attended the hospital, under the care of Dr. Garrod, for wasting; at that time it weighed twelve pounds (the normal weight being between twenty and twenty-one pounds). It rather rapidly gained weight, so that six months later it weighed twenty pounds. I saw the child about a year later, and the history then obtained was to the effect that the child had been getting very fat for the previous twelve months. It was unable to stand and talk. It had frequent "jumps," but no fits. The child was placid and contented, and you will notice now that the child is flat and flabby, with coldness and marked blueness of the extremities. Its head measures twenty inches, and is neither in excess

Digitized by Google

nor below the average. Many of these children exhibit vaso-motor disturbance. The child is mentally extremely deficient. There is very little actual rigidity of the limbs, and no spasticity of movement. The knee-jerks are, however, very active. Ankle-clonus is present, and there is a tendency to extension with the plantar reflex. During the past year very little alteration has taken place in the child; it has neither gained nor lost weight, and there has been no improvement in its mental condition. This case is shown more especially to illustrate the vaso-motor disturbance, which is often observed in these children.

CASE 5.—The next case is that of a girl fourteen years of age, and illustrates an advanced stage of the disease. She was seen for the first time some four years ago, and the history then obtained was that up to six months previously she had been a normal child. She could read, write, and do sums. She was probably backward, from the fact that she was at the age of ten only in the first standard at the board school. At this time she began to lose the power of doing her sums, then of writing and reading, and then weakness of the legs was noticed, though she could still walk fairly well when first seen. The legs were rigid, the knee-jerks were exaggerated, and ankle-clonus was present. The child began to get very fat, and to have a stupid, vacant expression. She talked slowly and somewhat indistinctly, and another curious fact was that she developed a great desire for money, which she kept closely guarded in a purse. The appetite was large. The next event was the occurrence of fits, the first being very slight and while attending Sunday school, and seemed to have affected the left side of the body only; she rapidly recovered from this attack. Shortly afterwards another fit occurred on the right side, and for a time she lost her speech; these fits have recurred periodically since. From that time onwards the disease has pursued a progressive course. The power of walking went. The arms and legs became rigid and flexed, and extremely wasted. The speech was entirely lost, but vision has been retained unimpaired, and she still recognises people. She exhibits very well the marked condition seen in the later stages of the disease—the arms flexed and strongly adducted to the sides, clenched hands, and the finger driven into the palms, the child being in the cramped attitude, occupying as little space as possible. The child has that difficulty in swallowing that is seen in so many of these cases of diplegia; liquids are fairly well swallowed, but solid food is taken only with difficulty. There is no lordosis or kyphosis. The head is not much bent forward. There is marked rigidity of the abdominal muscles, giving rise to the deformity of the chest. The whining cry of the child is quite typical. She still pays some attention to her surroundings. The legs are very wasted, and the knee-jerks are with difficulty obtained, owing to the rigidity. The big toe is "cocked" back with the typical extensor response.

CASE 6.—The next case is an example of diplegia following an acute illness. The child, who is now two years of age, was up till eight months old perfectly well. The child then had a fit and became blind, and after that the head was retracted and the back arched, so that the heels nearly touched the back of the head. The child became extremely wasted. When the child was about two years old she began to gain weight, but has never been able to sit up. The child at present is small for its age, but is very fat and flabby. The head is large and globular, being somewhat hydrocephalic. The legs are rigid and extended, and there is little or no power; the knee-jerks are increased and the plantar reflex gives an extensor response. Ankle-clonus is present. This is a case of acquired disease, and I think there is little doubt that the child suffered from an acute attack of posterior basic meningitis, from which it recovered, leaving, however, considerable damage to the cerebral hemispheres.

Morbid Anatomy.—The most usual condition met with and described in these cases of cerebral diplegia, is one of atrophy of the convolutions; this condition may be limited to certain regions of the cortex, or may be general. In some parts of the cortex it is so altered that it has a wash-leather appearance, and in some cases a cystic condition extends beneath the cortex known as "porencephaly." These cysts are generally situated between the cortex and the lateral ventricles of the brain, and do not usually open into the latter. They do not occur in the cerebellum, but a condition corresponding to the "wash-leather" appearance of the cerebral cortex is seen, the grey matter of the cerebellar cortex being extremely atrophied. Hydrocephalus is not infrequently found, but apart from changes in the cortex it

Digitized by Google

cannot be considered as a direct cause of the disease. Another condition found is that of thickening of the pia-arachnoid membrane, giving rise to and associated with atrophy of the cortex. This condition is usually met with in older children, and is always the result of congenital syphilis. As has been stated above, the most usual condition met with is atrophy of the convolutions, and the question arises, To what is this atrophy due? Various views have been held, and that which for a long time held the first place was the view that the condition was due to meningeal hæmorrhage produced by injury at the time of birth. Various facts go to show that this is not the most important factor. Another suggestion is that the condition is due to embolism, and there is certainly evidence to show that the localised atrophy is so accounted for; but this, again, is probably but a rare cause.

Thrombosis of the longitudinal sinus has been shown to be a cause of diffuse atrophy of the cortex, and it seems not unlikely that this is the cause of the porencephaly which occurs in these cases. Intra-cerebral hæmorrhage in the form of multiple hæmorrhages, as seen in asphyxia neonatorum, is also probably a cause. But although all these may be a cause of diplegia, yet it is probable that they are so in but a small proportion of the cases, and the view that the cerebral degeneration is due to some toxic process acting on the nervous system, either pre-natally or in early life, is the one against which least can be said. The nature of such a toxin is at present unknown, but considering the frequency with which the disease occurs in connection with congenital syphilis, it is extremely probable that that, at any rate, is one of the poisons; the occurrence of the disease after other acute specific fevers seems to make it probable that these supply other toxic agents. Even assuming that the condition is due to a toxin, one is still uncertain whether such a poison acts on the neuron or on the vascular supply, or possibly on both, by first impairing the vitality of the neuron by deficient blood-supply, and then acting directly on the neuron. It is certain that all cases cannot be attributed to the same cause. All the factors above enumerated have their value, and one judges of the lesion from the course which the case runs; for instance, in those cases which are slowly and steadily progressive it is reasonable to suppose that some toxin is at work; while in those in which the disease is stationary or retrogressive, it is reasonable to suppose that the condition is due to some past affection, which is not now in operation. The cases of family spastic paraplegia raise some very interesting points, for they do not seem to be accounted for by any of the above causes. The onset of the disease at a time of life when the functions of the limbs are called into activity, suggests that the condition of the neuron is only capable of performing a limited amount of work, and that under increased exertion it breaks down; or, to state the same thing in other words, its vital capacity is too small.

A few words must be said with regard to the microscopical appearance of the nervous system in these cases. In the cortex there is considerable degeneration of the various layers of the cells, and especially of the large pyramidal cells; passing down the pyramidal tract, one finds either absence, old degeneration, or recent degeneration in the course of the pyramidal fibres, and in the cord both the direct and crossed pyramidal tracts are degenerate.

Treatment.—In many cases the condition is slowly progressive, and no treatment is of avail. In other cases by regulating exercises a good deal can be done to prevent deformity and to help the child to walk. The go-cart also helps the child considerably. Tenotomy is advisable in some cases. Mental training in some cases is also important, and is attended with results that could hardly be expected.

THE CLINICAL AND PATHOLOGICAL RELATIONS OF THE CHRONIC RHEUMATIC AND RHEUMATOID AFFECTIONS TO ACUTE INFECTIVE RHEUMATISM.

Discussion* by the Chelsea Clinical Society.

WILLIAM EWART, M.D., F.R.C.P., in the Chair.

THE CHAIRMAN:—I much regret to state that Dr. Bannatyne, who should have been present here to-night, is prevented from attending. He has sent an abstract of the remarks he had intended to make, but it may be of advantage to postpone its perusal till our next meeting, when it is hoped Dr. Bannatyne may be able to speak.

My first duty in connection with this debate is to express the thanks of the Society, and their

* Held at the Jenner Institute, March 12th and 19th.

Digitized by Google

appreciation, for the hospitality which is tendered to them by the Jenner Institute.

The Chelsea Clinical Society, in its selection of a subject for last year's debate, was guided by a feeling that whereas a great deal had already been done in the cause of phthisis, the other national malady, rheumatism, which in some of its phases is extremely fatal, was not receiving the amount of attention which for so long it had deserved. Bearing in mind the clinical character of this Society, it was thought desirable to reverse the usual sequence, and to deal first with questions of treatment, and particularly with the treatment of the cardiac manifestations of rheumatism. This year the Council has again selected rheumatism, but this time in connection with its clinical and pathological problems. In medical thought and in medical language there is much uncertainty as to the relative scope of the two sets of affections. The boundary between true rheumatism and that which is pseudo-rheumatic or merely rheumatoid has yet to be drawn.

I will now call upon Dr. Archibald E. Garrod to open the debate.

Dr. ARCHIBALD E. GARROD, after discussing the Nomenclature and Classification of Rheumatism and Rheumatoid Arthritis, proceeded to say: I take it that the main question which we are invited to discuss this evening is whether or no rheumatoid arthritis, or, as I should prefer to say, any of the diseases included under that name, stands in any direct relationship to acute rheumatism. The existence of such a relationship has been warmly maintained by some, and equally warmly contested by others. Doubtless we have representatives of both camps among us this evening, and we may rely upon full justice being done to the arguments in favour of either view.

If such a direct relationship exists, we should expect the onset of rheumatoid arthritis to be frequently preceded by an acute or subacute rheumatic attack. It is true that a history of antecedent rheumatic fever, recent or remote, is not very infrequently forthcoming in such cases, as must inevitably be the case with two maladies both of which are so common.

Moreover the onset of rheumatoid arthritis in its more acute forms may be attended by a considerable amount of febrile disturbance, and at this stage it may be mistaken for rheumatic fever, especially as the initial febrile stage may be followed by a temporary improvement. I believe that this accounts for some of the cases in which a history of recent acute rheumatism is obtained.

An undoubted attack of rheumatic fever may be followed by a chronic joint affection, clinically indistinguishable from the fusiform variety of rheumatoid arthritis. A similar result may follow an attack of gonorrhœal rheumatism, or even gout, and the question arises whether such secondary rheumatoid arthritis is merely a continuation of the former malady, or affords an example of a second disease developing in a subject weakened by the antecedent attack, just as tuberculosis may appear as a sequel of measles. Such cases constitute a very small proportion of the total number. In the great majority of instances rheumatoid arthritis of any form appears as a primary malady, and presents its characteristic features from the onset; and more often than not the patient has not previously suffered from any troubles which are suggestive of a truly rheumatic origin.

Next, as to the development of abarticular rheumatic manifestations in the course of rheumatoid arthritis. In this connection it should be borne in mind that rheumatoid arthritis in all its forms is most apt to appear at a period of life at which the abarticular manifestations of true rheumatism tend to be less conspicuous. Some of these are almost confined to childhood and puberty, and even endocarditis and pericarditis diminish notably in frequency as age advances. Organic cardiac murmurs are undoubtedly present in some cases, and some published statistics place their frequency unexpectedly high. Some such murmurs clearly date from attacks of acute rheumatism at some period more or less remote, others may be ascribed to atheromatous changes. Each individual example requires to be considered upon its merits; and impressions based upon observation and experience are of at least equal value with statistics. Such experience as I have had leads me to range myself with those who hold that sufferers from rheumatoid arthritis show no greater liability to valvular disease than others not so afflicted, and I imagine that few medical men when watching such a case anticipate, as a probable event, the development of organic heart disease in its course. In children with so-called rheumatoid arthritis, Dr. Still has almost constantly

Digitized by Google

found pericardial adhesions *post mortem*, but the active pericarditis in which such adhesions presumably have their origin has passed unnoticed.

Subcutaneous nodules, which bear a striking resemblance to those met with in rheumatic children, are occasionally present in cases of rheumatoid arthritis. Those which I have seen have not had the transitory existence of the small deposits of lymph in the subcutaneous tissues of rheumatic patients, and it must not be forgotten that at the period of life at which they have been observed true rheumatic nodules are practically unknown, being confined to childhood and early adult life.

The question is one which can only be settled by pathological means, such as bacteriological examination, which, in the case of rheumatic nodules, has already yielded most interesting results in the hands of Dr. F. J. Poynton and Dr. A. Paine.

Indeed, it is quite possible that before long bacteriology may throw such light upon the whole problem of the relation of rheumatoid arthritis to rheumatism as will render further discussion of the question futile. Meanwhile it appears to me that the evidence adduced in support of the common causation of rheumatism, and any variety of rheumatoid arthritis, is not of such a nature as to carry conviction.

If rheumatoid arthritis is not a rheumatic affection, what then is its pathology? Clearly this question can receive no satisfactory answer until we know for certain whether we are dealing with one disease or several diseases. It is quite possible, and even probable, that the several forms differ entirely in their pathology, and that a theory which is true for one form may be wholly incorrect as far as another is concerned. The nodular or senile cases, with their slow progress and essentially chronic characters, present many features which suggest that the lesion present is of the nature of a degenerative or trophic change in the joints, comparable to the degenerative changes met with in other structures. Such a view of its nature receives support from the striking resemblance of the articular lesions here seen to those met with in the joints of sufferers from nervous diseases such as locomotor ataxy and syringomyelia. In these cases, at any rate, there is nothing to suggest a rheumatic origin, and it is in these that the effects of mechanical influences are most marked.

We shall have the advantage of hearing Dr. Bannatyne, whose name is so closely associated with the bacteriological investigation of the disease under discussion, and who, in collaboration with Drs. Wohlmann and Blaxall, has isolated a definite bacillus from the synovial fluid in a number of cases—a bacillus which they have altogether failed to find in the joints of sufferers from other forms of articular disease. On clinical grounds we should expect that, granting that certain cases of rheumatoid arthritis are infective, the cases concerned would be those of what have been spoken of as the fusiform type. In the similar affection met with in children, enlargement of the spleen and lymphatic glands is frequently observed, and it is in such cases that the constitutional symptoms tend to be most pronounced.

Among the predisposing causes of the more acute cases debilitating influences take the first place; the onset may follow upon a period of ill-health and attendant lowered vitality, and of late years an attack, or a series of attacks, of influenza has often appeared to be the predisposing cause of the onset, or of the re-awakening of the disease from a dormant condition.

The relation of rheumatoid arthritis to gout is another subject of considerable interest and not a little difficulty. Sufferers from chronic gout may exhibit joint changes, which, in their clinical features, bear a close resesemblance to those of rheumatoid arthritis of the nodular kind,—as, for example, the enlargement of the terminal joints of the fingers known as Heberden's nodes. On the other hand, I can see no valid ground for the belief that gout plays any part in the causation of the more acute forms.

There is certainly much room still for the accumulation of facts, both clinical and pathological, bearing upon the nature of rheumatoid arthritis.

Let us first make sure that when we employ this term, or any other terms which may eventually replace it, we are referring to some one definite morbid condition, and not to a congeries of diseases only superficially alike. Then by the diligent piling up of fact upon fact, a task which will provide employment alike for the clinician, the bacteriologist, and the morbid anatomist, we may hope to construct a foundation so solid that

Digitized by Google

the superstructure of theory raised upon it will be capable of resisting any strain to which it may be subjected.

In the present state of our knowledge one is justified in taking up an expectant attitude in this matter; and I, for my part, feel certain of one thing only, which is, that I know far less about the pathology of rheumatoid arthritis than I thought I did ten years ago.

The Chairman then invited Dr. F. J. Poynton to read the following communication:

The Infectivity of Acute Rheumatism, with Especial Reference to the Chronic Types of the Disease.

By F. J. Poynton, M.D., M.R.C.P.,
Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and

Alexander Paine, M.D.,
Assistant Bacteriologist to St. Mary's Hospital.

When we undertook this investigation into the bacteriology of rheumatic fever, the view that acute rheumatism was microbic in origin had been almost established by the labours of many investigators, both in this country and on the Continent. So, at the outset, we had the advantage of this strong support, and, in addition, the advantage of the experience of previous investigators upon the question of the bacteriology. But, although advances had been made, the bacteriology of the disease was still a complicated problem, because of the various results that had been obtained.

These, for example, were the various questions that suggested themselves:

(1) Was there a specific organism? If so, was it a bacillus or micrococcus?

(2) Was the disease the result of a mixed infection?

(3) Was it a result of the attenuation of the pus-forming staphylococci and streptococci?

(4) Was it a result of many different infections? In other words, is acute rheumatism a peculiar phase of many different diseases?

Again—

(5) Does the organism remain localised to one spot, or is it widely diffused through the system?

We fell back upon the teaching of clinical and pathological medicine as our fixed point, from which to make a start, and first considered this problem:

Is acute rheumatism, as we recognise it in England, a definite disease?

The answer we believe must be in the affirmative. It is possible that clinicians may have drawn too rigid a line around it in some directions, and may have confused allied conditions in others, but the mean result of many years of experience has, we think, established acute rheumatism as a definite disease with definite peculiarities.

If this be granted, then if one micro-organism be constantly found to be present, and when isolated be found to produce identical lesions in animals, it is most probable that this organism is specific.

We do not propose to enter into the difficult question of "mixed infection," further than to say that in our experience acute rheumatism is probably the result of infection by one micro-organism, and to criticise some statements that are occasionally made upon this view of mixed infection.

It has been said that in rheumatic fever, pericarditis, nodule formation, pleurisy, and so on are due to secondary infection; whereas rheumatic fever, with swollen joints which react to salicylates, is the true disease, due to some infective agent as yet unknown. If then pericarditis and nodules develop in a case of joint rheumatism, these latter are looked upon as the result of secondary infections.

This appears to us untenable for two reasons. The first is, that pericarditis, pleurisy, and nodules are clinically and pathologically manifestations of the disease, and not superadded phenomena; and therefore to regard polyarthritis as the criterion of rheumatic fever, and the other affections as complications, is not in accord with our present knowledge of the disease.

The second reason is that though salicylates are no doubt of the greatest practical value in some cases of acute rheumatism, we are not justified from the standpoint of strictly scientific medicine in considering them as tests of the rheumatic nature of the disease. We know practically nothing of the chemistry of rheumatic fever, and not much more of the action of salicylates within the body; therefore to make the reaction to salicylates a proof of rheumatism, and the failure to react a proof that the disease is not rheumatic, may lead to error.

Digitized by Google

Those, then, who claim that acute rheumatism with severe visceral inflammation is a mixed infection, because the disease in the adult often shows itself as a polyarthritis reacting to salicylates, claim, we think, more than is legitimate. They have a right to demand an explanation of the action of salicylates in these cases, for the subsidence of the rheumatic process under their use must have some meaning, but, in our opinion, cannot claim what may be a phase of rheumatism as a distinct disease.

Those who consider acute virulent rheumatism as due to a mixed infection, because they cannot find organisms in the simple arthritic type, but can do so in the virulent type, have more reason for their belief; but this position we believe we have considerably weakened by demonstrating that the organisms in acute rheumatism are rapidly destroyed at the sites of the lesions, and are therefore not easy to demonstrate in the tissues, more especially if the attack be not severe.

Again, the diplococcus may be very difficult to find in the arthritic exudation, though it may be present in considerable numbers in the synovial membrane.

How emphatically the clinical study of acute rheumatism points to the infective origin of the disease is very clearly put in the following sentences from Dr. W. B. Cheadle's introduction to the discussion on acute rheumatism at the annual meeting of the British Medical Association in 1895:

> "The occasional epidemic prevalence, the variability of type, the incidence upon the young, the concurrence of tonsillitis, of endocarditis, of pericarditis, of pleurisy, of pneumonia, of erythematous eruptions: the rapid anæmia, the tendency to capillary hæmorrhage and albuminuria: the implication of the joints, the relapses, the occasional supervention of hyperpyrexias, the nervous disturbance, the specific power of salicylic acid, are all suggestive of an infectious disease."

We have now isolated from fourteen cases of rheumatic fever a diplococcus which grows in liquid media in streptococcal chains, and on solid media in staphylococcal masses. Triboulet before, and Wasserman and Malkoff during the time we were working, had also isolated a diplococcus from acute rheumatism. This year also Fritz Meyer has published the results of a prolonged investigation of the bacteriology of rheumatic angina faucism, and has also isolated a diplococcus which in many respects, especially in the experimental results it produces, resembles the one we have described, and with which we have obtained similar results.

Our researches have been more detailed than those of others, and though different media have been used by various workers, it seems very probable that the organism in each case is the same. Thus, for example, though Meyer used a medium of high alkalescence, and we used a faintly acid medium, in both instances the organism grew well on blood-agar, which is only faintly alkaline. We have isolated the organism three times from the blood of patients suffering from rheumatic pericarditis, twice from the throat, four times from the pericardium (growing them also in the exudation), twice from the urine, three times from the cardiac valves, and once from the rheumatic nodule. Intra-venous inoculations into rabbits have produced valvulitis, pericarditis, fatty changes in the myocardium, polyarthritis, nodular formations, pleurisy, and choreiform movements. The urine, alkaline in the rabbit in health, becomes acid and loaded with urates. In no instance has there been suppuration in the viscera. The anatomy of the lesions in the rabbit is identical with that in man. We have demonstrated them in man in the visceral and parietal pericardium, cardiac valves, pleural and peritoneal exudation, tonsils, rheumatic nodules, and in the rheumatic joint, in the rabbit in the cardiac valves, pericardium, joint exudations, kidneys, liver, connective tissues, pia mater, and cerebro-spinal fluid.

We were indebted to Mr. H. G. Plimmer, Pathologist to St. Mary's Hospital, for the inoculations, and for much valuable advice and criticism. All the cases that were chosen were cases of undoubted rheumatic fever, not a shadow of doubt as to their nature entering the minds of the physicians to whom we were indebted for the generous loan of the notes. When we mention the names of Dr. W. B. Cheadle, Dr. D. B. Lees, Dr. F. G. Penrose, and Dr. A. E. Garrod, we feel that this will be a sufficient guarantee of the really rheumatic nature of our cases.

We have therefore concluded that this organism is a cause of rheumatic fever, for if we strip that disease of valvulitis, polyarthritis, pericarditis, pleurisy, nodules, and chorea, it is difficult to believe in its existence. There is, then, only left

Digitized by Google

the constitutional tendency, a factor of first importance, but not the disease itself. Further, we believe it to be the only cause, though more researches are needed to prove this point.

It is important to make one statement about the medium with which we obtained our most conclusive results. It consisted of milk and bouillon rendered slightly acid with lactic acid. Working in the dark, we made use of this medium, and adhered to it because of the results, but it must not be thought that we believe it to be necessarily the best—it was our best. Nor did we use lactic acid because of the lactic-acid theory of rheumatic fever, but because it was the best practical method of slightly acidifying the medium. The organism grows well on blood-agar, which is not an acid medium.

To demonstrate them in the human tissues is not easy, because they are rapidly destroyed at the sites of the lesions; they are small, and cling closely to the connective tissues, and since they rapidly decolourise by Gram's method—though they stain by it,—they are not easy to differentiate in the tissues.

If these observations are corroborated, the pathology of rheumatic fever is in a firmer position than heretofore, and when once the toxins can be isolated we shall be in a position to approach the explanation of the various lesions both acute and chronic, with more confidence than we can at present.

To turn now to certain points which perhaps bear more closely upon the subject under discussion.

We have repeatedly demonstrated the diplococcus in the joint exudate in rabbits, and have also found them in the joints in rheumatism. The organisms are rapidly taken up by endothelial cells and polynuclear leucocytes in these situations, and possibly this may account for the difficulty of demonstrating them in acute rheumatic arthritis, which is usually of less severe type than that produced experimentally in the rabbit. The presence of the organisms in this form of arthritis brings acute rheumatic affections of joints into line with arthritis, the result of septic, gonorrhœal, and pneumonococcic infections,—according to the researches of Drs. Bannatyne, Wohlmann, and Blaxall, rheumatoid arthritis must be included in this group also. Gouty and syphilitic arthritis remain as examples in which no micro-organism has been demonstrated, the former, perhaps, not being the result of a microbic infection, the latter almost certainly being micro-organic.

One joint in one of the rabbits that developed polyarthritis remained chronic, and did not subside. In every other instance the swellings either completely subsided, or the animal died from the infection; but in this case, though the animal recovered except for some slight valvulitis, the left shoulder-joint remained swollen and slightly tender from May 30th until October 8th, when the animal was killed. Then we found much gelatinous swelling of the connective tissues around the joint, with thickening of the capsule and erosion of the cartilages. This result suggests that researches in this direction may eventually throw light upon the more chronic forms of rheumatic arthritis.

Another point to which we wish to call attention is the condition of the kidneys in acute rheumatism. In fatal cases we have found very definite changes in the parenchyma, especially of the convoluted tubules. There is marked cloudy swelling of the protoplasm of the renal cells with fatty change, and some desquamation. The change is diffuse—that is, it affects a great number of the convoluted tubules. Further, we have isolated the diplococcus from the urine in acute rheumatic pericarditis.*

In rabbits, possibly because of their fur, the kidneys are more severely affected, but the changes are of the same type; there is much cloudy swelling and also some desquamation of the renal cells, and in their kidneys we have repeatedly demonstrated the micro-organisms. Further, in the rabbit we have traced this condition step by step to the formation of a white infarct, which certainly in the rabbit, and most probably in man, is *not* the result of a gross embolism, but probably the result of the invasion of a considerable number of the diplococci, by the blood-stream, to the particular area. As a result of this invasion, the cloudy swelling in that area passes over the limit to the stage of necrosis, and thus is produced a white infarct.

It appears to us that the kidney is an important site for elimination of the poisons produced by

* Recently we have isolated the diplococcus from the kidney and urine in the bladder after death.

the rheumatic infection, and that in this process the kidney itself to some extent suffers. Usually the injury is not gross, for though albuminuria, and in some malignant cases hæmaturia and nephritis occur in this disease, as a rule there is not albuminuria or evidence of serious renal inflammation.

When a white infarct heals, we know the result is a scar, and it is possible that repeated attacks of acute rheumatism may do permanent damage to the renal tubules, not of the gross nature found in the infarct, but of a grade sufficient in severity to destroy the functions of many tubules, and perhaps lead to the gradual destruction of the renal cells, and their replacement by fibrous tissue. In brief, repeated attacks of acute rheumatism in the young may lead to fibrosis of the kidney in the adult, or, short of that, impair the function of the kidney for the elimination of the rheumatic poison in later life. As a result of this it may be that a subsequent attack of rheumatism in adult life—though less severe, because the adult is less susceptible—is more chronic, because the poisons are eliminated with more difficulty.

When we turn to gout we know that the kidneys suffer much in the disease, and that some authorities give to the kidneys the first place in its causation, and all allow the impairment of their function as a prominent factor in the more chronic, inveterate, and cachectic types.

For these reasons it seems to us important to study the kidneys very closely in acute rheumatism, and perhaps it may be found that albuminuria is more frequent than is generally supposed, or that the peculiar condition of cyclical albuminuria may sometimes be a result of the impairment rather than destruction of function that appears to be the result of rheumatic fever.

The last point we touch upon is concerned with malignant endocarditis. We have now collected many facts to show that some of these cases are truly rheumatic, and not the result of secondary infections.

This suggests that some of these lesions of joints that run on to ulceration and destruction of cartilage are really rheumatic. The disease has altered in type, and for some reason has become locally malignant in these situations, as it sometimes becomes in the valves. This we should say is no new suggestion, but is commented upon in the classical work upon pathology by Wilks and Moxon.

The Chairman then called on Mr. Macnamara to read his paper.

The Toxic Origin of Rheumatoid Arthritis.

By N. C. Macnamara, F.R.C.S.,

Consulting Surgeon, Westminster Hospital.

I seem like Rip Van Winkle, roused from a long sleep by your very kind and flattering invitation. I have been asked to say a few words with reference to the toxic origin of rheumatoid arthritis, and I will endeavour as shortly as possible to explain the reasons which have led me to consider that a certain number of cases presenting symptoms and pathological changes characteristic of this form of disease have originated in the action of chemical substances produced by specific micro-organisms. In the first place, it is clear that the poisons produced by specific micro-organisms in some cases cause rapid necrosis of tissues, as, for instance, pyæmic suppuration of joints, anthrax, erysipelas, and so on; then we have a class of cases such as those caused by the tubercle bacillus, running sometimes a rapid course with necrosis of the pulmonary or other tissues; in others, as in tubercular diseases of the articulations, often following a chronic course, and terminating in anchylosis and deformed joints. Lastly, there is an essentially chronic class of cases depending on the poisons produced by micro-organisms such as leprosy; midway between these chronic and the more acute forms of disease, depending on toxic poisons, we have an intermediate class of which gonorrhœal rheumatism affords us a typical example, and one which bears directly on the subject now under discussion. We had long ago suspected that the arthritis in these cases was the result (in persons peculiarly susceptible to such influences) of the absorption of the products of the urethral suppuration into the circulation. But it was not until the year 1879 that it was proved by cultivations that the gonococcus was the cause of the urethritis; and let me here remark that because this is an undoubted fact, we by no means conclude that every case of chronic or of acute inflammation of the urethra depends on the gonococcus. This specific form of micro-organism has been found in the submucous tissues of the

Digitized by Google

urethra long after the acute symptoms have ceased; and what is very much to the point, it has also been found in the synovial tissues, and in the sheaths of tendons around joints affected by gonorrhœal rheumatism. In the mucous membrane the coccus in the first place causes a suppurative inflammation, but in the synovial tissues a subacute, non-suppurative form of inflammation. And it would seem that this chronic arthritis is largely sustained by fresh infection from the urethra, for until this latter is cured, the gonorrhœal rheumatism will continue in spite of all treatment. In one of the most persistent cases of this form of arthritis I ever met with the patient contracted influenza and pneumonia, from which he died. On making a post-mortem examination we found gonococci in the tissues round some of the affected joints; osteophytes had sprouted out from the end of the bones, and the root-like processes of dense fibrous tissue connected with the insertions of the tendons were found projecting into the cancellous tissue of the bone, a pathological condition which, so far as my experience goes, is only found in cases of rheumatoid arthritis.

Passing from gonorrhœal rheumatism, we naturally come to the class of cases described by Dr. Todd, as far back as 1854, in which symptoms of chronic rheumatoid arthritis were attributable, in his opinion, to the absorption from the uterus of vitiated secretions, or, as we should call it, septic materials. I well remember a case of this kind in Dr. Todd's wards, and the clinical lecture he published on it. The patient was a young woman with her wrists, fingers, and other joints fixed and deformed in a manner characteristic of chronic rheumatoid arthritis. In the year 1885, I was asked by Dr. Donkin to see a woman, twenty-five years of age, then in his wards (she is still in the incurable ward of the Westminster Hospital), who presented typical symptoms of chronic rheumatoid arthritis. She had been in service; there was no history of rheumatism or of gout in her family; her uterine functions were normal, and Dr. Donkin was unable to discover any satisfactory cause for her symptoms, many of her joints being deformed and fixed. Her breath was extremely fœtid, and she had extensive dental caries, to which, in my opinion, her symptoms were attributable. Under proper treatment her mouth was placed in a healthy condition, and then her long-continued symptoms of rheumatism disappeared, her joints of course remaining fixed and deformed. Dr. W. Ewart has lately published ('Brit. Med. Journ.,' September, 1900) a case of malignant endocarditis, which he is disposed to think originated from infective matter, absorbed, as he suggests, from the sockets of carious teeth. For some fifteen years I have watched cases of chronic arthritis, and although I know that some of them have followed acute rheumatism, others being attributable to gout, and some, so far as one could form an opinion, to neurosis, still I am convinced that a certain number of these cases are due to toxic poisoning, and that no treatment in such cases is of any lasting benefit until the source of infection has been removed.

I have referred to dental caries especially because it is a disease from which so many people suffer; nevertheless, as so comparatively few of this number are affected with rheumatoid arthritis, it seems evident that not many of us are susceptible to the deleterious action of the toxic poison to which I have referred. But the conviction has slowly become fixed in my mind that in certain cases definite toxic poisons are the cause of the symptoms and the pathological changes characteristic of chronic rheumatoid arthritis. I cannot help thinking that it is possible some of us hardly realise the fact that this form of joint disease is not always due to rheumatism, but may be caused by gout, by toxic poisoning, or it may be due to a neurosis,—conditions which require divers forms of treatment, especially in their early stages, if we are to hope for success in preventing or relieving the symptoms to which they give rise.

This has been the upshot of my experience, and, as I say, Dr. Ewart kindly asked me to come here and refer to the matter. No doubt much advance has been made, as we have heard this evening, with regard to these forms of rheumatoid affections, especially of the joints, and one cannot help believing that in cases such as those to which I have referred, some of our younger members will be able to isolate, and to show us the micro organism which I am quite convinced in a considerable number of these cases is the root and origin of the disease.

The Chairman then called on Dr. C. O. Hawthorne for his contribution to the discussion.

Digitized by Google

The Relationship of Acute Rheumatism to Chronic Rheumatoid Arthritis.

By C. O. HAWTHORNE, M.D., M.R.C.P.

The point in this discussion to which the Council has suggested I should address myself is the relationship of acute rheumatism to chronic rheumatoid arthritis, and in proceeding to consider that relationship, I think it is well at the outset that we should define exactly those points concerning which there is general agreement. We are, for example, all agreed that there is an acute disease characterised by general febrile disturbance, and by pain and swelling affecting several or many joints, and this disease we call acute rheumatism or rheumatic fever. We are all agreed that the severity of the attacks of rheumatism may vary very widely, so that we allow the possibility of a subacute rheumatism the attacks of which, I presume, nobody doubts are essentially of the same nature as the condition we call rheumatic fever. Further, we are all at one upon the fact that there is a condition, for the most part chronic in its course, and marked by swelling and deformity of many joints, which we are in the habit of calling chronic polyarticular rheumatoid arthritis, and the question at issue is, Is chronic rheumatoid arthritis simply a chronic form of the disease which we know as rheumatic fever, or is it an entirely different disease? To that question there are completely opposing answers offered. We are told by some authorities that chronic rheumatoid arthritis is simply rheumatism; that, in short, there exists a series of gradations commencing with acute rheumatism, and passing through subacute rheumatism, to a chronic deformity of the joints, namely, chronic rheumatoid arthritis. On the other hand, we have a statement to the effect that whatever chronic rheumatoid arthritis may prove to be, it is certainly not a rheumatic arthritis. The position at the outset, therefore, presents itself as one of affirmation on the one hand, and denial on the other. But when we proceed a little further we find that this is not exactly the position. We find that whilst there are those who hold that chronic rheumatoid arthritis is simply rheumatism, the strength of the opposition to that theory lies mainly in the erection of opposing theories. There is the theory which is especially associated with the name of Dr. Bannatyne, that chronic rheumatoid arthritis is a disease which depends upon micro-organisms. There is the theory to which we have just listened, that the condition is due to septic absorption. Upon these suggestions I do not venture to offer an opinion. They have the force which attaches to the distinguished authorities who uphold them, but they have hardly attracted any large body of supporters.

It is the dystrophic theory of rheumatoid arthritis which has been most strongly pressed, the theory that the phenomena of rheumatoid arthritis depend upon disturbances in the trophic nervous apparatus, these disturbances being either primary changes in the spinal cord, or changes induced in the spinal cord by irritation reflected from the pelvic or other viscera. I propose to try and examine some of the facts which bear upon this controversy. And I would remark, in the first place, that it is allowed in the experience of all physicians that a not inconsiderable number of cases of chronic rheumatoid arthritis are found in patients who have at some previous period in their lives suffered from typical acute or subacute rheumatic attacks. Indeed, there are instances, not so very unusual, in which the chronic rheumatoid condition follows immediately upon an attack of acute rheumatic fever, so much so that the rheumatoid condition, as it were, grows out of the rheumatic fever, and it is impossible to select any point of time and to say that here rheumatism ends, and here rheumatoid arthritis commences. Again, if the family histories of these patients are examined you will find, I believe not infrequently, that there are distinct evidences of rheumatism in its generally admitted forms in members of the family immediately related to the patient. You find a middle-aged patient with rheumatoid arthritis, whilst a brother or sister is the subject of chronic valvular disease obviously the result of acute or subacute attacks of rheumatism which have been recognised in early life. Again, there are certain cases in which the symptoms, the history, and the physical facts are such that it is impossible to exercise any confident judgment, and to say this is a case of rheumatism, not rheumatoid arthritis, or this is rheumatoid arthritis and not rheumatism.

When from clinical experience I find that the rheumatoid condition sometimes immediately grows out of an attack of rheumatism, that the two

Digitized by Google

conditions display themselves in different members of the same family, and are sometimes commingled in one and the same patient, I am inclined to pause for some considerable time before I am prepared to say that rheumatoid arthritis is a condition which has nothing at all to do with rheumatism.

I pass now to consider other evidence. One of the most confident grounds on which an endeavour has been made to mark a distinction between rheumatism and rheumatoid arthritis, is the absence from the latter of cardiac disease. Endocarditis we all recognise to be in the great majority of instances an evidence of rheumatism. But it must be remembered, and Dr. Garrod has reminded us of the fact already, that it is an expression of rheumatism which is specially prone to manifest itself in childhood and early adult life. It is by no means a common expression of rheumatism in middle life. But the majority of patients who suffer from chronic rheumatoid arthritis are patients who are approaching or have reached middle age, and therefore it by no means follows that the absence of cardiac complications from the great majority of cases of rheumatoid arthritis is conclusive evidence that rheumatoid arthritis is a condition which is quite distinct from rheumatism. But is it the fact that cardiac complications do not occur in rheumatoid arthritis? The great French physician, Charcot, made a statement in exactly the opposite direction. His teaching, as no doubt you know, was to the effect that chronic rheumatoid arthritis is simply a form of chronic rheumatism, nodular rheumatism as he preferred to call it; and he states from his unrivalled experience that cardiac lesions occur fairly frequently in cases of rheumatoid arthritis. Experience in the same direction was also provided by Trousseau, who notes the occurrence in nodular rheumatism of occasional inflammatory lesions of the pericardium. For my own part I am quite ready to admit that I have never been able to satisfy myself of the evolution of valvular disease during the study of an individual case of rheumatoid arthritis.

Let us look at the question of subcutaneous nodules. Until quite recently we were taught that subcutaneous nodules were pathognomonic of rheumatism. Later experience has shown that similar nodules undoubtedly emerge during the progress of a certain number of cases of rheumatoid arthritis. It is quite true that in most instances the nodules of rheumatoid arthritis are larger and much more persistent than the ordinary so-called typical rheumatic nodule which appears in childhood and young adult life. But if we bear in mind the extremely chronic and obstinate character of the great majority of cases of chronic rheumatoid arthritis, it is not by any means surprising that subcutaneous nodules, when they occur, should assume a more persistent character than subcutaneous nodules do in the comparatively fleeting attacks of rheumatism which come on in childhood and early adult life.

The fact that in rheumatoid arthritis we get nodules essentially similar to those which were claimed as distinctive of rheumatism, seems to me a very suggestive one. I do not wish to press it too far, because it has been shown that similar nodules also appear in gout and in various other conditions, and therefore the argument to that extent is qualified. Still, I put it to you as an important consideration, that a condition, namely the eruption of subcutaneous fibrous nodules, which was claimed with great confidence as being a distinctive and an infallible note of rheumatism, has been found in recent years to appear also in chronic rheumatoid arthritis.

I now look at the development of muscular atrophies in chronic rheumatoid arthritis. Upon the face of it, of course, the development of muscular atrophy, such as we see so frequently and to such marked extent in chronic rheumatoid arthritis, does seem at first sight to afford strong presumptive proof of the accuracy of the teaching that the phenomena of rheumatoid arthritis depend upon changes in the central nervous system. We know that changes in the trophic nervous system often produce muscular atrophies, and that certain lesions in the spinal cord are competent to produce severe articular disturbances. Hence it is not unnatural to argue from the development of muscular atrophy in rheumatoid arthritis that all the phenomena of the disease depend upon trophic nervous change. I wish to put it to you that an entirely different view is possible. It was taught by Sir James Paget many years ago—and as far as I know, his teaching is not disputed,—that any joint lesion may be followed by atrophy affecting the muscles which act upon that joint. The joint

Digitized by Google

lesion may be very slight, and yet the muscular atrophy which follows may be very considerable. The atrophy is certainly not due to disuse; it is too rapid and extreme to be accounted for in that way. It picks out particular groups of muscles and neglects others, and therefore there is no issue upon the question of disuse. But the suggestion is, that the joint lesion, through afferent nerves leads to trophic disturbances in the centres of the spinal cord, and that these disturbances are prejudicial to the vitality and to the nutrition of the muscle. Now if it is the case that a slight joint lesion, say a sprain, is competent to produce muscular atrophy it follows, of course, that other lesions in joints are competent to produce muscular atrophy, and as a matter of fact such muscular atrophies may sometimes be observed in the course of acute rheumatism. Now if it is the case that the occurrence of muscular atrophy following a sprain does not prove that the original joint lesion depended upon trophic disturbances in the spinal cord—and nobody, of course, is prepared to argue that it necessarily follows that the occurrence of muscular atrophy in cases of chronic rheumatoid arthritis is no proof at all that the articular changes of that disease depend upon central changes in the spinal cord,—the sequence of events after a sprain is presumably irritation of the joint, disturbance of trophic centres through afferent nerves, resulting in muscular atrophy. If that sequence of events is true in the case of a sprain, it is at least possibly also true in the case of chronic rheumatoid arthritis. Instead of concluding, therefore, that because we have muscular atrophy in cases of rheumatoid arthritis the articular disturbances of the disease certainly depend on the nervous change, the more reasonable assumption, or at least an equally reasonable assumption is that the first changes are changes which take place in the joints, that from these there follow disturbances in the spinal cord, as a result of which there develop muscular atrophies.

Considering, then, all these different lines of evidence, and considering also the fact that pathological anatomy has been unable to produce for us any demonstration of the existence of changes in the central nervous system competent to produce the articular and other changes of chronic rheumatoid arthritis, I am loath to commit myself to a definite opinion that chronic rheumatoid arthritis is due to changes in the trophic nervous apparatus. On the other hand, I am not prepared to go so far as to say that I hold very strongly that rheumatoid arthritis is certainly rheumatism; at least, I am not prepared to go so far in the absence of definite proof that the same ætiological factor is concerned in the two processes. We appear to be on the very eve of knowing confidently and scientifically what is the ætiological factor of acute rheumatism, and when that is known we shall be in a more reasonable position to find out whether rheumatoid arthritis, and other conditions which have been more or less frequently recognised as rheumatic, depend upon the same cause or different causes; but for the great majority of us at present the term rheumatism is simply a symptom-complex. When we speak of rheumatic fever we mean that a patient is feverish, and has swollen and painful joints and other symptoms to which I need not allude. We do not suggest that we are dealing with a recognised and definite causative agent.

It may be said that to decline to pledge a definite opinion in favour of either the rheumatic or the dystrophic theory of rheumatoid arthritis is to halt between two opinions. I do not think the charge is a very serious one. What has been called the cross-bench type of mind is not popular amongst politicians, but it can hardly be urged as reproach in medical circles, where so little is known, so much is vague and uncertain. Theories come and theories go, sometimes they go even more readily than they came. I am not suggesting that they have not their value and use, but they have also their disadvantages, and one of the disadvantages is that they tempt us to leave the secure ground of the impartial clinical investigation, and to become advocates and partisans. I submit there is no reason at all why we should pin ourselves definitely to a theory as to the relationship between chronic rheumatoid arthritis and rheumatism, and in the present position of the evidence the safe and judicious and logical position is the cultivation of the open mind, and the only justifiable verdict is that of "not proven."

The Chairman:—The society is to be congratulated on the work which has been done here to-night, and particularly on the demonstration of the organisms of acute rheumatism by Drs. Poynton and Paine. It is not for me to offer any remarks upon the statements and the opinions which have been offered, but merely to tender the thanks of the Society to those who have provided us with so much excellent food for thought, and to express a hope that we may all be able to listen to the important contributions which have been promised for our next meeting.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 446. WEDNESDAY, MAY 15, 1901. Vol. XVIII. No. 4.

CONTENTS.

PAGE

*With Mr. Watson Cheyne at King's College Hospital 49

*Some Remarks on the Relationship between Rheumatism and Rheumatoid Arthritis. By GILBERT A. BANNATYNE, M.D., M.R.C.P. 53

*Discussion on the Clinical and Pathological Relations of the Chronic Rheumatic and Rheumatoid Affections to Acute Infective Rheumatism. By—

R. TANNER HEWLETT, M.D. 54

W. ARBUTHNOT LANE, M.S., F.R.C.S... 55

WM. ARMSTRONG, M.R.C.S. 56

ALEXANDER PAINE, M.D. 62

DR. BLANC (Aix-les-Bains) 62

ARCHIBALD E. GARROD, M.D. ... 63

*With Mr. Ballance at St. Thomas's Hospital 64

** Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, etc., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications, with the exception of those relating to Advertisements, should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 1s. 6d. each, or will be forwarded post free on receipt of 1s. 9d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

WITH MR. WATSON CHEYNE AT KING'S COLLEGE HOSPITAL.

MARCH 4th, 1901.

Coxa vara.—This boy, æt. 15, presents an interesting case from the point of view of diagnosis. The left leg is slightly shorter than the right, the left being thirty-six and three quarter inches and the right thirty-seven and one eighth. There is slight adduction and limited abduction. There is also restricted flexion and defective internal rotation on the left side; there was a good deal of pain at first, but that has passed off now. What does that collection of symptoms mean? It is not a tuberculous hip-joint; if it were I should not be able to roll the limb about so freely as I can, especially in a case where considerable restriction of movement exists. The limb would be too painful, and further, it would be quite rigid. It is a case of coxa vara, or curvature of the neck of the femur, a condition which occurs sometimes in adolescents, but most frequently in young children. When the disease was first described, it was noticed only in adolescents; but since then one has found many more cases in infants, with curvature very marked. The most frequent form of curvature of the neck of the femur is a double one, with the convexity forwards and upwards. The shortening is due to the curve of the femur; and the defect in abduction, which I have, however, only seen in cases in young adults, is, no doubt, due to a considerable extent to the hitching of the curved neck against the acetabulum. You may have other curves of the neck of the femur in this condition, so the term "curvature of the neck of the femur" is now becoming generally adopted rather than the term coxa vara, for the latter is strictly applicable only when the curvature is outwards. In children the movements are generally more free than in adolescents. You will often find that children can put their legs round their neck, and external rotation is generally very free. In adults there is more or less restriction of

Digitized by Google

movement, and in adolescents at the commencement of the condition there is pain. But in the case of children, the patients are not brought because of pain, but because they cannot walk properly, and the feet are turned out.

With regard to the causation, in children, no doubt, it is rickets. Rickets, as you know, is the most common cause of softening of the bones. I should think that in adults also rickets is at the bottom of the disease, although in some cases no other signs of rickets can be found; at any rate it is something which causes a softening of the neck of the bone and consequent curvature. There is little doubt that it is the weight of the body on the softened bones which causes the curvature. That being so, as soon as you see the condition coming on you must take the weight of the body off the legs. That can be done by putting a patten on the other foot, and making the patient get about with crutches. In children the condition is usually bilateral; it has been so in all the cases in children which I have seen. In the case of this boy, however, it is more pronounced on one side than on the other; still, I think it is commencing in the other leg.

With regard to treatment, the first thing is to take the patient off his legs; there are various forms of apparatus which are made suitable for such cases. I do not think this boy requires anything done in the way of operation, because the deformity is comparatively slight. But where the deformity is bad you must do something. There are two forms of treatment which are suitable for particular cases. One, which is especially intended for adults, is taking out a wedge of bone from the neck of the femur in the trochanteric region. This attacks the deformity directly. The base of this wedge is so arranged that when the edges of the gap are brought together the bones come back to their proper position. I believe the parts are too small in children to get a satisfactory result from the wedge method. What I have done in the case of children is to saw across the femur below the trochanters, hold the trochanter as far forward as it will come, and then take the loose lower limb and rotate it inwards until it is in the normal position of extreme internal rotation. Then I nail on an aluminium plate with tin tacks on both fragments, so that when the trochanter is as far forward as it can come, the foot is in the normal position of extreme inversion. The result as regards walking has been extremely satisfactory, and further, what I did not expect, the growth of the limb on the side operated on has been much improved. In two cases in which I operated only on one leg, I found after some years that the limb operated on was a good deal longer than the other. One case I saw four years after operation, and found that the leg I had operated on was two and a half inches longer than the one I did not operate on. The correct way to put it is that the leg I did nothing to did not grow as long as it should have done by two and a half inches. The leg being in proper position, and the movements being corrected, the leg had gone on growing normally, whereas the leg which had not been rectified, and was therefore incapacitated, did not grow so well. That is difficult to explain. I divided the femur above the entrance of the nutrient artery, and I therefore diminished the vascularity of the neck of the femur. Increased vascularity might have had something to do with the softening of the bone. The operation not only restored the limb to its proper position, but enabled it to grow properly, whereas the leg not operated upon did not grow: in future, therefore, I shall operate on both legs at the same time.

Dupuytren's contraction.—This case is one of Dupuytren's contraction of the palmar fascia. He comes partly for this condition of his palm, and partly on account of a painful swelling on the other hand. The swelling came on suddenly without cause three days ago, with severe pain and marked redness of the skin; the swelling and redness are now passing off, leaving the skin a little discoloured. In fact, he has had an attack of gout in the other hand. Dupuytren's contraction is also said to be essentially due to gout. It usually commences towards the ulnar side of the palm, and as the bands of fascia going to the fingers contract, the ring and little fingers, and at a later period the middle and forefingers also, are gradually flexed till they may actually touch the palm. The tendons are not contracted.

As regards treatment, there are three chief methods: one is tenotomy, introducing the tenotomy knife underneath the skin, and nicking all the tight bands of fascia in every direction, until the fingers can be got straight. The knife must, as a rule, be introduced in various places, and all the bands

Digitized by Google

must be thoroughly divided. There is no doubt that in cases where the skin is not much shortened much improvement often follows the employment of tenotomy, but after a time the condition tends to recur. Another plan, which might be carried out in this case, is to dissect out the bands of fascia. Here we have a single band with radiating branches, and if that band is dissected out, and the skin brought together over the wound, the case will do well. A third method, which is useful in some cases, is to divide the skin and fascia in a V-shaped manner, with the point of the V towards the arm, and then to straighten the finger. The two sides of the V are then stitched together, and the apex is attached to the two sides at the distal part. None of these operations is good for all cases, and in some a combination, especially of tenotomy with the V-shaped division, will be the most useful. This is especially the case where the fingers are much flexed into the palm. I think in this case we should excise the piece of dense skin with the fascia beneath it, and stitch the sides of the wound together.

Inoperable scirrhus of the breast.—This is a woman of only thirty years of age, who comes with a recurrence of cancer in the breast. She has had trouble in the breast for two years, and at the commencement of it she consulted a doctor who said that it was nothing. Last June she went to one of the London hospitals, where the old operation for removal of the breast was performed. A month later she went back to the same hospital, and some glands were removed from the axilla. Now, six months afterwards, she comes here with the condition which you see. The remains of the breast are everywhere converted into a hard mass, which is adherent to the skin. The skin is red; the redness extends almost to the umbilicus, and round the ribs and up to the axilla. It looks at first sight almost like a spreading erysipelas. Numerous nodules are also present n the skin, over this red area; in fact, it is a case of commencing scirrhus *en cuirasse*.

She has also a lump in the other breast, large masses in the axilla, and numerous glandular nodules in the neck. She has been rapidly getting weaker, and has not been able to do any work for two or three weeks. She also has a bad cough. She is very anxious to have something done, but any attempt to remove the disease is of course out of the question. I am, however, rather tempted in this case to remove her ovaries. I have not cured any patient by that means, but I have seen remarkable improvement as an immediate result. What I have found—and it is the experience of others also—is that marked improvement follows in the first instance, but that after from six to eighteen months the effect disappears. I cannot explain this result. It seems to me as if removal of the ovaries takes away something which is essential for the life of these cells, or which is at all events of great importance to them, and that after some time some other organ furnishes the secretion, and thus makes up for the deficiency, and enables the cancer to grow again. The use of thyroid extract has also been recommended, but I have not seen any improvement from it; it is usual, however, to administer it in cases where the ovaries have been removed.

Mr. Stanley Boyd has had one case where freedom from recurrence was of much longer duration. One of the best cases I have had was operated on two years ago. A patient came with a very similar condition, very advanced, with at the same time profuse menorrhagia from a large fibroid. I felt that she was going downhill as rapidly as she could, and so I thought we would stop the bleeding by taking away her ovaries. The effect of that was an atrophy of the fibroid, cessation of bleeding, and almost complete disappearance of the malignant disease. In that case I took away as much of the malignant disease as I could, but I did not try to get beyond it. I only took away the greater part of the mass. That case was still well last summer, except that there were one or two glands in the neck, which, indeed, had never quite disappeared. But she came back to me in January, and the condition looks as if it is relapsing. In another case, which I saw eighteen months ago, the ovaries were removed, and I believe that all the visible disease disappeared. I saw her, however, again in January of this year with the whole condition practically as bad as ever.

[In this case the ovaries were removed, and the patient recovered from the operation quite well, but so far the disease has apparently been quite unaffected, in fact it has spread very considerably since the operation.]

Flatfoot.—Here is a young woman, æt. 24, who complains of pain on the inner side of the feet and

Digitized by Google

in the ankle, and she says it is getting worse. Both feet are flat, the great toes are everted, and she has commencing bunions. There is no difficulty in restoring the arch. The treatment here will consist of suitable exercises, more especially what are called tip-toe exercises. When the patient stands on tip-toe she raises the arch of the foot. Other exercises are rising on tiptoe, bending out the knees, and sinking down on the outer side of the feet, also turning out the feet and standing on the outer side. These exercises should be done two or three times a day. The patient must also be instructed to walk with the toes pointing forward. In people with flatfoot the tendency is to walk with the feet turned out. It is also desirable to support the arch by means of a spring which comes forward as far as the bases of the toes, and backwards to the great tuberosity of the os calcis. Her boot should also have the instep strengthened. The hallux valgus is very slight, and a toe-post is all that will be necessary for that. The cases of flatfoot in which operative treatment is required are those where you cannot restore the arch of the foot, and where the patient is suffering great pain. Pain alone is not a sufficient indication, because you may have an acute development of flatfoot, which improves if the foot is put up in plaster of Paris in the corrected position. But where in addition to pain you have a fixation of bones, no exercises will undo it, and you may have to operate and restore the arch. There are a great many operations for restoring the arch. One of the simplest is to remove the head of the astragalus, and put up the foot in plaster of Paris for some weeks in the corrected position. Some take a wedge out of the neck of the astragalus; others take a wedge out of the tarsus, etc. But I believe the most satisfactory operation, as a general rule, is removal of the head of the astragalus.

Movable kidney.—This patient is a woman æt. 39. There is nothing noteworthy in the family history or in her own previous history. Early in the summer of 1900 she noticed she was passing water more freely than usual, though not much at each act. A fortnight later she began to feel a stabbing pain in the right loin, which was tender to the touch, the pain when it came on lasting very acutely for about an hour. It was accompanied by intense desire to pass water, and relief had to be obtained at once. After a time the pain began to shoot down from the right loin to the symphysis pubis. The trouble is limited to the right side. The patient has been losing weight. The various organs are healthy. The urine is normal; sp. gr. 1115; no pus or blood or deposit of any kind. There never has been any blood in the urine, nor has the patient ever noticed anything abnormal about it. The right kidney is very freely movable, in fact it is a floating kidney; the left kidney cannot be felt and is not tender. The diagnosis here is not quite clear. The history suggests stone in the kidney or tuberculous kidney, but the normal condition of the urine and the absence of bleeding is against either of these troubles; on the other hand, these are not quite the symptoms to which a movable kidney alone would give rise. It might be a case of movable kidney with intermittent hydronephrosis, but in that case the pain is not so continuous, nor is there the same frequency and urgency of micturition; usually there are intervals of complete freedom from pain. I should not like to give a positive diagnosis, but I think we shall find something more than a mere floating kidney. As her symptoms are severe, I propose to cut down on the kidney and investigate its condition. If it is merely a floating kidney nephrorrhaphy is, I believe, the best treatment. However, I shall bring the kidney out of the wound in the loin and examine it thoroughly before stitching it.

[On exposing the kidney it was found to be the seat of acute miliary tuberculosis with a breaking-down deposit about the centre, close to, but not opening into, one of the calyces. The kidney was excised, and the patient was completely relieved and left the hospital in three weeks.]

THE MEDICAL GOLF TOURNAMENT will be held this year on the links of the Mid-Surrey Golf Club, Richmond (kindly lent for the occasion), on Thursday, June 6th. The competition will consist of 11 holes *v.* Bogey—the usual division of competitors into Senior and Junior being made. First and second prizes and a prize for best last 9 holes *v.* Bogey will be given in each class. Foursome sweepstakes will be played in the afternoon. Entries stating lowest handicap, and enclosing 5*s.* to be sent on or before June 5th to the Hon. Sec., Medical Golf Tournament, Rosedene, Windlesham, Surrey. It is proposed to hold a dinner in London on the same evening if a sufficient number of men promise to attend. Tickets 7*s.* 6*d.* exclusive of wine.

Digitized by Google

THE CLINICAL AND PATHOLOGICAL RELATIONS OF THE CHRONIC RHEUMATIC AND RHEUMATOID AFFECTIONS TO INFECTIVE RHEUMATISM.

Discussion * by the Chelsea Clinical Society.

WILLIAM EWART, M.D., F.R.C.P., in the chair.

In the absence of Dr. Bannatyne, the Chairman called on the Hon. Secretary to read the following communication:

Some Remarks on the Relationship between Acute Infective Rheumatism and Rheumatoid Arthritis.

By Gilbert A. Bannatyne, M.D.Glasg., M.R.C.P. Edin., Hon. Physician Royal United and Royal Mineral Water Hospitals, Bath.

The study of the relationships of diseases of the rheumatic group opens up a very wide field, which, to adequately cover, requires more time than is at present available, so to-night I will confine myself to a few observations with regard to those forms of disease—acute infective rheumatism, and rheumatoid arthritis. Clinically these diseases in their acute and typical forms are quite separate and easily distinguishable from one another, and perhaps from this aspect and in this stage they have more points of dissimilarity than any other two diseases of the rheumatic group, but we cannot say the same for certain and more uncommon stages of their development.

An ordinary case of acute infective rheumatism is characterised by a sudden onset, by a rise in temperature until it often reaches a high range, by joint pains and joint swellings, by heat and redness of the affected joints, by sour-smelling perspirations, and by cardiac lesions. It is an acute disease running an acute course, is readily influenced by the salicylates, and tends in the majority of cases to clear up, leaving little or no permanent joint destruction. On the other hand, rheumatoid arthritis in the acute form begins more gradually in one joint, probably one of the phalangeal ones, but spreads rapidly to practically all the joints, and affects often the temporo-maxillary joint. The joints of the fingers present a characteristic spindle-shaped swelling, and the rise in temperature, although marked, seldom shows a range as high as is seen in acute infective rheumatism. The extremities are cold and sweating, and show a livid mottling, while numerous small freckles or patches of pigment appear on the skin of the face, forearm, or hand, while other vaso-motor or trophic changes are common. Muscular atrophy is marked, whilst the glands proximal to the joint are often enlarged. Cardiac changes are more uncommon than in acute infective rheumatism. Rheumatoid arthritis is further characterised by a progressive course, for once it attacks a patient it tends to get worse constantly, by exacerbations, which, however, get less and less acute; but unfortunately the patients usually become more and more crippled, until in the long run they are quite helpless. Once seen, these two diseases can never be confused, and I hold they are separate and individual disorders, and not merely varieties of the same disease. But what can be said about those more rare cases which commence apparently as moderately acute attacks of infective rheumatism, but which refuse to respond to treatment, and which, as time passes, gradually become more and more distinctive of rheumatoid arthritis? And again, how can we class those cases which, after several attacks of acute infective rheumatism, develop rheumatoid disease? It is these cases which give rise to trouble in diagnosis and prognosis. Are they *mixed* infections, or does the rheumatoid become engrafted on the infective rheumatism? My theory is that these are cases of rheumatoid arthritis engrafted on an attack of acute infective rheumatism. Anything which damages or weakens the resistance of the body generally, but more especially of the joints, is bound to lay the latter open to the attacks of the rheumatoid poison. Statistics show that rheumatoid arthritis frequently follows other diseases of the joints, such as gout, or gonorrhœal arthritis, as well, of course, as rheumatic arthritis. I do not look on this as showing that these diseases have any specific action in the causation of rheumatoid arthritis, but rather that they simply act as injuries to the joint structures, and thus render them more liable to the invasion of the rheumatoid poison, by diminishing their resistive powers and lowering their vitality. Statistics do not show that

* Held at the Jenner Institute, March 12th and 19th.

Digitized by Google

rheumatoid arthritis follows acute infective rheumatism with much greater frequency than it does any other joint trouble, and the clinical history of the disease would hardly lead one to suppose that it was merely a variety of acute rheumatism, or one which with any certainty followed such an acute attack. So the two poisons seem to be something different and quite distinct. Rheumatoid disease does not possess the characters either of a degeneration; reference to its clinical symptoms again shows us that. Of course I am here speaking only of the acute forms of rheumatoid arthritis, for the chronic varieties follow altogether a different course, and might well be secondary degeneration following on any acute joint trouble. These differences are so marked, that it makes me doubt whether the two states (acute and chronic) have really any relationship one to another. This must be clearly understood to follow my line of argument. Pathologically there seems little proof as to the nature of the diseases whose clinical appearances can be so readily recognised. In acute infective rheumatism Drs. Poynton and Paine have found a micro-organism which may well be the specific cause, but which they wisely claim only to be one of the causes. This, I think, is wise, in so far as our present knowledge is very elementary, and it may be that two or more organisms have the power of producing symptoms which are clinically indistinguishable. While I say this is possible, I do not look on it as probable; but proof will shortly, no doubt, be forthcoming. The same may well be said of rheumatoid arthritis. Dr. Wohlmann and I found a micro-organism in one form of this disease, but it may be found, on further investigation, that other microbes also have the power of producing rheumatoid symptoms. The micro-organism found by Drs. Poynton and Paine differs from that found by Dr. Wohlmann and myself in characters and life-history—one being a micrococcus, and the other a diplobacillus. Recently I have examined the fluid from several cases of well-marked rheumatoid arthritis, and have not succeeded in isolating Drs. Poynton and Paine's micrococcus. This is purely of negative value. My opinion at present, therefore, is that the two diseases as seen in the acute stages are quite separate and distinct, and that the only relationship between them is that, as a predisposing cause to rheumatoid arthritis, acute infective rheumatism may play an important part in weakening the resistance and vitality of the body and joints in a certain small proportion of cases; but that it plays a more important part than influenza, tonsillitis, or gonorrhœal rheumatism in its causation I do not believe. The hereditary tendency to joint troubles is marked in rheumatoid disease, but it may be that it is gout, not rheumatism, that is inherited. In a family with arthritic inheritance, one member may develop gout, another rheumatism, while yet a third rheumatoid arthritis. Rheumatoid tendencies most frequently affect the females of an arthritic family; but that these inheritances and their results show more than a weakened joint-resistive power there is nothing to show.

The Chairman: After hearing the paper, I am sure we all feel the greater regret that the author was not able to be here to deliver his views himself. It will be noticed that Dr. Bannatyne is clear that rheumatoid arthritis and acute infective rheumatism are two separate affections, even though they may sometimes be associated at their origin. I will now call on Dr. Hewlett to address the meeting.

Dr. Tanner Hewlett: Mr. Chairman and gentlemen, although I am called upon to address you this evening, I feel that I could better play the *rôle* of listener and learner in this most important debate, and therefore my remarks will be brief. With regard to the bacteriology of rheumatism and rheumatic affections I am afraid that I have very little practical experience. In the only case of acute infective rheumatism from which I had the opportunity of examining the fluid and the inflamed joint, strangely enough I isolated a bacillus which, in all its characters, resembled the *Achalme bacillus*, but, of course, on a single case one does not base any conclusions. With regard to Dr. Poynton's and Dr. Paine's organism, I congratulate them heartily on having isolated it; I think it is undoubtedly a new organism, and therefore the discovery of it is very suggestive, though I think they would be the first to admit that further investigation is required before it can be definitely said that this organism is the specific cause of acute infective rheumatism. Unfortunately the cases which one can examine are so few and far between, that it is difficult to get sufficient material, at any rate in any short time, to produce absolute proof that an organism is the cause of acute infective rheumatism. With regard to rheumatoid arthritis, I had the opportunity of

Digitized by Google

seeing a good deal of Dr. Blaxall's work, and it seemed to be very careful; and unquestionably, I think, the organism which he isolated is quite distinct from the organism of Dr. Poynton and Dr. Paine.

Coming next to the clinical side of this subject, in the first place, it seems to me that the terms rheumatism and rheumatoid are very largely a survival of the ideas of our forefathers. The old physicians applied the terms rheumatism and rheumatoid to conditions involving the joints, the idea being that some acrid humour flowed to these parts, hence the term rheumatism. I think we see this idea in the various ways in which the term is used,—rheumatism, chronic rheumatism, rheumatoid arthritis, gout, rheumatic gout, and gonorrhœal rheumatism, terms all involving the use of rheumatic or rheumatoid, but which are applied to very different conditions. Dr. Bannatyne has somewhat cut the ground from under my feet, for I was going to express very much the same opinion that he did, namely, that acute infective rheumatism and rheumatoid arthritis are really quite distinct diseases, and I believe they will be found to be due to different ætiological agents. Taking the two diseases as a whole, there are certainly some differences in their symptoms, and it seems to me that later on we shall find they are certainly distinct. We have to remember that identity of clinical symptoms, and nearly identical pathological lesions do not necessarily prove identity in ætiology. A very good example of that may be seen in the case of sore throat and membranous conditions of the throat. Without a very careful examination by bacteriological methods, it is practically impossible in many instances to say what the particular lesion of the throat may be due to, so we must be very careful in drawing conclusions simply from apparent identity in clinical symptoms or pathological lesions. Unquestionably toxic conditions—the toxæmias—for some reason or another, have a great predilection for the joints. We find that the joints are affected in a large number of toxic conditions, and I may mention a few of them in addition to rheumatism and gout. We get arthritis in scarlet fever, influenza, dysentery, yaws, verruga permana, and especially in Malta fever. Then of course the infective agent in pyæmia is especially carried to the joints, and lastly, there is that remarkable toxic arthritis which we get after the injection of antitoxic serum. All these cases show that toxic conditions of the blood have an especial predilection for the joints. I may definitely say, then, that I agree with Dr. Bannatyne that acute infective rheumatism and rheumatoid arthritis are probably quite distinct diseases. Coming to the cases in which a good deal of difficulty arises—cases of the rheumatoid condition, arthritis deformans supervening on acute infective rheumatism, there again I agree with him. It appears to me that in all probability the rheumatoid condition is grafted on to the rheumatic one, and it is merely a coincidence that the one condition occurs following after the other. As I say, I believe we have been swayed by tradition, and if we could have a committee of experts who had to rename these conditions, if they had all the evidence before them, I think they would state that rheumatoid arthritis is a different disease from acute infective rheumatism, —that is to say, that it is a disease due to different ætiological agents.

Mr. W. Arbuthnot Lane said: When your Secretary did me the honour of asking me to take part in the very interesting but equally vague discussion which forms the subject of this debate, I attempted to crystallise what little information I thought I might possess about it. The chief difficulty which I experienced was the want of definition of the terms chronic rheumatic and rheumatoid affections, since they comprehend such very different conditions in the minds of various observers. I determined therefore to confine my remarks entirely to facts of which I have some slight knowledge, however unimportant may be their share in the groups denoted by the above-mentioned terms.

I find from experience that the term chronic rheumatic affection of a joint is used to comprise conditions of joints in which there is pain of a rheumatic character unaccompanied by any marked change in the anatomy of the joint, except perhaps some effusion into it. This so-called rheumatic pain is of a most indefinite character. For instance, it has on many occasions fallen to my lot to remove a damaged internal semilunar fibro-cartilage from a knee-joint, and to restore to the articulation its normal painless functions in cases that have been diagnosed as chronic rheumatic affections, and treated as such without success. This is only one of many instances, and I relate it not to criticise the treatment employed, but merely to illustrate the fact that a condition of the knee-

Digitized by Google

joint which to one observer suggests the presence of a chronic rheumatic affection because of recurring attacks of pain, limited movement, and effusion, to another appears as a typical traumatic condition.

Therefore the most marked feature of chronic rheumatic affections of joints is pain, often variable in amount, and influenced greatly by the weather, etc. Now if this is the case, there are innumerable traumatic or mechanical conditions which exhibit precisely these symptoms, and which I see usually diagnosed and recognised as chronic rheumatic, or if there are distinct joint changes, as rheumatoid. I would at once exclude from consideration the true rheumatoid arthritis, which we all now know as a definite specific disease, varying in type within wide limits in the several decades of life, and having no relation whatever with acute rheumatism.

When I was first interested in the so-called rheumatoid arthritis, or osteo-arthritis, I found the terms were used synonymously to comprise all pathological joint conditions which were not distinctly tubercular. The student was taught, and for all I know to the contrary is probably still taught, that these terms denote a disease of a joint which commences in the articular cartilage, evidenced first by a change in it, and later by its removal. There followed on this an eburnation and destructive change in the exposed bone, and a new osseous formation on the margins of the articular surfaces. Pieces of bone or osteophytes were developed in the synovial fringes and ligaments, and in some forms of joints the mechanics were so altered that an excessive movement of the bones on one another took place, while in others they became united to one another by buttresses of bone. The student was expected to enumerate a sequence of this sort as the pathological evidence of this so-called disease, "chronic rheumatoid or osteo-arthritis." A careful examination of these pathological conditions showed me that there was no such disease whatever, but that these terms comprised a large number of conditions or variations in the anatomy of the skeleton produced by traumatism, applied on one or on a few occasions, or by some alterations in the mechanism of the part due to excessive function or to specialisation of function.

It is more than fourteen years ago since I first published my observations on the subject in the 'Transactions' of the Pathological Society, and all my subsequent experience and observation on the subject have only served to verify my earlier views. These conditions have no relation whatever to acute rheumatism, or, as far as I know, to any chronic rheumatic affection; while, on the other hand, their causation is most clear and precise, and purely and simply mechanical. The very few minutes at my disposal make it impossible for me to do more than touch on the merest fringe of a subject which is to me full of the greatest interest, not only as bearing on alteration in the anatomy and physiology of the individual, involving frequently great pain and functional inactivity, but also as underlying the whole problem of evolution. Personally I regard the terms "chronic rheumatic" and "rheumatoid" affections (apart from true rheumatoid arthritis) as unmeaning terms, and used too often as useful cloaks for ignorance. It is quite time we were a little more precise in our knowledge of the causation of the several joint conditions which are so common. I do not fancy that much general improvement will arise till we have become more familiar with the physiology of the skeleton, and its behaviour under varying mechanical influences.

Mr. Wm. Armstrong (Buxton) said: When your Council was good enough to ask me to take part in this most interesting debate, it expressed the wish that I should deal more especially with the ætiology of chronic rheumatic and rheumatoid affections.

Much as I admire the bacteriological work recently done by Drs. Poynton and Paine, I must at once say that I cannot accept their conclusions that micro-organisms are the sole factors in the causation of those cases which, characterised by fever, pain, and swelling of the joints, and by endo- or pericardial mischief, we group together under the name of acute rheumatism.

The man who whenever he gets his feet or clothes damped almost immediately feels stiffness behind the angle of the jaw, followed, if this be not arrested, by tonsillitis and rheumatism, with or without fever, cannot to my mind be looked upon as suffering from the effects of a specific germ; nor yet the one who on the advent of a north-east wind finds that his liver becomes inactive and his stools pale in colour, and who knows from experience that if he does not deal with this condition promptly, tonsillitis and following rheumatism will probably be his fate.

Digitized by Google

Expert evidence, both bacteriological and clinical, certainly points to the fact that there is a specific form of acute rheumatism, but I do not think that our present information is sufficient to justify us in putting *all* acute rheumatic cases down to the credit of this factor. But we are not so immediately concerned this evening with the causation of acute rheumatism as with the influence of this ailment upon chronic rheumatic and rheumatoid affections.

Dr. Archibald Garrod in his very able paper opening this discussion says, "The main question to be discussed this evening is whether or not rheumatoid arthritis (or as I should prefer to say, any of the diseases included under that name) stand in any direct relationship to acute rheumatism." But I think we may go further than this, and consider chronic rheumatism and gonorrhœal rheumatism in addition to the various forms of rheumatoid arthritis.

I turned to the paper of Drs. Poynton and Paine in the hope of distinct enlightenment, as its title "The Infectivity of Acute Rheumatism, with especial reference to the Chronic Types of the Disease," seemed to promise definite food for thought; but I was much disappointed to find that the only references to chronic conditions were the following:

(1) That they had repeatedly found the diplococcus in the joint-exudate in rabbits, and had also found it in the joints in rheumatism; but they do not say whether these cases were acute or chronic.

(2) That in gouty or syphilitic arthritis no micro-organism had been demonstrated, and that in the former type they were most probably not present.

(3) That one joint in a rabbit remained swollen for eight days, and then, when the animal was killed, much gelatinous swelling of the connective tissue round the joint was found, with thickening of the capsule and erosion of cartilages.

I submit that this does not carry the case for the infective origin of chronic rheumatic and rheumatoid affections much further.

We might now turn for a moment to *chronic rheumatism*. Very few of the sufferers from this ailment have ever had acute rheumatism; most of the cases seem to have come on from the effects of damp or exposure; and often in people living on clay soils, who get immediate benefit on removal to more suitable districts. With the exception of those cases in which a subacute affection of the joints follows an acute attack, I have rarely been able to connect those chronic manifestations of rheumatism with the acute form of the disease.

Gonorrhœal rheumatism undoubtedly holds a place of its own. I call it rheumatism rather than arthritis, because it not only attacks joints, but also tendons, ligaments, muscular tissues, and some of the structures of the eye.

Undoubtedly it is a toxic condition due to absorption from the urethra and deposition of the gonococcus in the tissues. On two important clinical points gonorrhœal rheumatism differs from rheumatoid arthritis:

(1) The absence of that severe wearing "tooth-achy" pain so characteristic of arthritis, such pain being quite independent of movement.

(2) The very small amount of muscular atrophy present even after many months of severe joint trouble.

Ord has suggested that in addition to the toxic factor there is a neural one, caused by irritation conveyed to the spinal centres through the agency of the nerves of the prostatic urethra, and I have seen indications that seemed to support that view; but in any case I am sure that this influence is a comparatively small one. It is rare to have much rise of temperature in gonorrhœal rheumatism excepting when acute pain comes on in the intercostal muscles.

I feel certain that although gonorrhœal rheumatism may appear in a rheumatic subject, and its course may thus be somewhat influenced, there is no actual connection between the two diseases.

Rheumatoid arthritis, however, is on an entirely different basis, and a careful study of nearly fifteen hundred cases has made me hold somewhat definite opinions.

Dr. Garrod sounds the key-note to this discussion when he warns us against the tendency to assume that all joint affections are necessarily rheumatic or gouty in origin, and he makes also a most important observation when he expresses his belief that under the name of rheumatoid arthritis are confused together several distinct forms of articular disease.

Acute arthritis differs in many important par-

Digitized by Google

ticulars from acute rheumatism. The temperature rises markedly at night. There is a very rapid pulse, like that which occurs in Graves's disease, its rate often being from 110 to 130 beats per minute. The perspiration never has the peculiar acid smell present in acute rheumatism, but the skin often gives off a most offensive odour, and the pain is of a peculiar "toothachy" character, and is often excessive. These cases frequently end in spasmodic contractures, muscular atrophy and distortion, and ankylosis of joints. As Dr. Garrod says, they almost always follow influenza or some other debilitating nerve influence.

CASE 1. *Acute arthritis following influenza.*—M. O—, female, æt. 17, always highly nervous and sensitive. Influenza early 1896, followed in three weeks by acute pain and effusion into wrists, knees, and elbows, quickly extending to other joints till hardly one joint was free. Seen end of 1897; pulse 110 to 130. Temperature never under 100°, at night often 101°; heavy perspirations of offensive smell; excessive pain of toothachy character in all joints; weak and emaciated. Strong spasmodic contractures and much muscular atrophy had come on during last three months.

Treatment.—Combined "Open-air" and Weir-Mitchell methods, D'Arsonval sinusoidal electric bath (200 alternations per second) being substituted for massage and electricity, Liq. Auri et Arsen. Bromid. in 5-drop doses three times daily. Slow but steady recovery took place, the spasmodic contractures of ankles, wrists, elbows, and fingers gradually relaxing. In July, 1898, the joints were broken down under anæsthesia; the left elbow was not movable; in one knee and one wrist the adhesions were very firm, in other joints less so. There was slight recontraction in the two joints named. This has now disappeared, and ankylosis of the left elbow-joint and inability to completely straighten two fingers of one hand and one of the other are the only signs of the disease remaining.

That one form of rheumatoid arthritis does sometimes follow acute rheumatism I am convinced, but I have never seen it come on unless the patient has been much debilitated or worried. The following case is typical:

CASE 2. *Acute rheumatism; nerve strain; rheumatoid arthritis.*—A. G—, female, æt. 25, had acute rheumatism at fourteen and eighteen years of age. Subjected to great nerve strain during the whole of 1897. January, 1898, sat for two hours in wet clothes; acute rheumatism in many joints, with pericarditis and endocarditis. After six weeks finger-joints became spindle-shaped, and the character of the pain altered, becoming "wearing and toothachy." June, 1898, came to Buxton; carpal joints much swollen, knees and feet painful and swollen, finger-joints spindle-shaped, loss of power in hands, marked muscular atrophy and debility.

Treatment.—Mild Buxton thermal and constant current electric baths alternately, and generous diet; drank Buxton thermal water; rapid improvement and complete recovery.

This type is quite distinct, is not very common, and is in no way like acute arthritis. I have notes of some twenty such cases, in all of which there was distinct evidence of acute rheumatism *plus* debility or anxiety. This is a very small proportion out of nearly 1500 cases. There are, of course, a considerable number of cases called by the name of rheuma*tic* arthritis, in which middle-aged or elderly patients, subjects of chronic rheumatism, but who have never had rheumatic fever, develop probably from mechanical or senile causes, arthritic conditions in various joints.

It is very possible in the earlier stages of acute rheumatoid arthritis to mistake it for acute rheumatism, but the difference as the case progresses is very marked.

Although I have gone into the matter most closely, I cannot confirm Dr. Hawthorne's observation, that there is in any considerable number of rheumatoid patients a personal or family history of rheumatism. Gout certainly forms the base of many of the slower cases of arthritis.

CASE 3. *Gout; great mental anxiety; arthritis; cardiac dilatation; albuminuria.*—H. S—, male, æt. 70 years. Gout for thirty-five years; for last ten years chronic gout in wrists, hands, and knees. February, 1898, after three months' great mental anxiety the character of the pain in the hands and knees changed, the carpal swellings increased, the interosseous muscles and those of balls of the thumbs quickly atrophied. August, 1898, came to Buxton very weak and ill, joints exquisitely tender, the "toothachy" pain in the wrists being especially trying. Cardiac dilatation and irregularity of pulse; some albumin in the urine but no casts;

Digitized by Google

excretion of uric acid low, only 65 per cent. of normal amount.

Treatment.—Drank Buxton thermal water. Baths being thought inadvisable, the affected joints were placed in the "Greville" superheated air apparatus. A modified "Salisbury" diet (much butcher's meat, and very little starchy food) was given, and two glasses daily of port wine matured in the wood. Improvement immediate; the excretion of uric acid was at once stimulated; nutrition improved; the cardiac and kidney conditions became better, and the joints lost their tenderness. Seen August, 1899, hardly any sign of joint mischief, and in very good general health.

Case 4. *Gout; nerve shock; arthritis.*—H. P—, male, æt. 58. Gout for ten years. July, 1898, sudden death of wife under most distressing circumstances; almost immediate increase of pain and swelling, and "uselessness" of wrists, hands, and shoulders, and pain in articulations of jaw. Buxton, February, 1899: looked weak and ill; much indican and calc. oxal. in urine. Excretion of uric acid below normal.

Drank Buxton thermal water. Had mineral and constant-current electric baths. Diet: much meat, but very small quantity of carbohydrates; port wine (matured in the wood). Marked improvement; excretion uric acid increased; pain lessened; nutrition improved. During treatment had attack of articular gout in left great toe; this was cleared away by three applications of the "Greville" superheated air. December, 1899, hardly any trace of disease remaining.

Mr. Macnamara's cases of arthritis from leucorrhœa and from decayed teeth respectively are most interesting; although I do not think that in either case the result was due to direct infection of the joints by means of the absorption of morbid material.

Ord pointed out how greatly certain tracts in the spinal cord were influenced by utero-ovarian irritation, and many cases are on record where arthritis lingered on until this source of irritation was eliminated.

Case 5. *Utero-ovarian trouble following confinement; arthritis.*—M. N—, female, æt. 27. Never had gout or rheumatism, nor any family history of these complaints. August, 1895, confinement followed by subinvolution, endometritis, menorrhagia, and ovaritis. October, 1895, arthritis of hands, shoulders, neck, jaws, and knees. In 1896 two courses of mineral baths abroad with no good result. In 1897 "superheated air" treatment. April, 1898, came to Buxton; very weak and ill; joints tender and painful; preliminary uterine treatment recommended but declined.

Had constant-current electric baths; slight improvement. September, 1898, returned to Buxton. Menorrhagia and ovarian tenderness worse than ever, and joints beginning to get more painful. Under pressure consent was obtained to local treatment. Uterus curetted and tampons of ichthyol used; then bath course similar to former one given. Rapid improvement took place. November, 1898, joints quite well.

The arthritis from decayed teeth is most interesting, but I doubt the direct absorption theory, and incline toward that of nerve-poisoning. I might quote a curious case in support of this contention.

Case 6.—E. F—, married, æt. 35, had suffered four years from gradually increasing cardiac irregularity; the heart was constantly going through all sorts of gymnastics. She had had two full courses at Nauheim without benefit, and was eventually sent to me for treatment by central galvanisation. Her teeth were in the condition described by Mr. Macnamara, and her breath was most offensive. As I could find no other cause of reflex irritation, and as the heart itself was healthy, I advised removal of the teeth before treatment commenced, thinking that these might be the reflex cause of the trouble. She declined the operation on the ground that anæsthesia would be dangerous, so I advised her to see Sir Douglas Powell, who supported my view. The teeth were all removed, and from that day to this (four years), there has never been the slightest cardiac irregularity.

This case is mentioned by Sir Douglas Powell in his book on 'Cardiac Neuroses.' The irritation in this case could not have come from the effects of direct absorption, but must have travelled by way of the nervous system; so it is not far-fetched to think that probably the patient mentioned by Mr. Macnamara on Tuesday last went through a similar process.

That other toxins are capable of causing arthritis is, I think, proved by the following cases.

Case 7. *Duodenal dyspepsia; arthritis.*—A. C—, male, æt. 56. Dyspepsia for years, discomfort

Digitized by Google

coming on from two to three hours after food. Early in 1897 pain and swelling in carpal joints, left shoulder and fingers of both hands, then in knees and ankles. Several baths courses, brine and Aix douche, without result; gradual progression of disease and deterioration of health. Seen October, 1898: much indican, skatol, cresol, and phenol in urine; aortic regurgitation. Baths being inadmissible, strict "Salisbury" diet was prescribed. General faradisation, the hand of the masseur being used as the labile electrode, was given daily. Betol, eight grains, one hour after each meal. Buxton thermal water drunk.

Steady improvement; joints became less painful and less swollen. Indican and allied bodies rapidly decreased. After six weeks' strict dietary was put on a modified dietary, which was kept up for six months, at the end of which time the joints were almost normal.

Case 8. *Duodenal fermentation: intestinal absorption: arthritis.*—F. C—, female, æt. 56, subject to secondary dyspepsia for years. Seen July, 1896; arthritis of almost every joint; very stout and flabby; cardiac action feeble; marked constipation; urine contained much indican and skatol, and an excess of the aromatic sulphates; had massage baths of short duration, with douche to spine; drank Buxton thermal water; methylene blue two grains, one hour after food, and a daily tonic aperient; meat diet; gradual steady improvement; loss of unhealthy fat; cardiac action stronger. Did well till March, 1897, when became careless as regards dietary and took much bread and sweet cake; two months later severe relapse. September, 1897, came again to Buxton, worse than at first as regards joint troubles; put on "Salisbury" dietary and constant-current electric baths. May, 1898, returned much improved. January, 1900, well, excepting slight swelling and stiffness of one knee.

Case 9. *Chronic constipation; arthritis.*—B. H—, female, æt. 46; most obstinate constipation for ten years; bowels moved with great difficulty and by enormous doses of strong aperient medicines; for five years gradually increasing swelling and stiffness of knees, wrists, ankles, jaws, and shoulders, with much neuritis, muscular atrophy, and loss of power.

Seen June, 1897; tongue thickly furred, breath most offensive; the urine showed a very large increase of aromatic sulphates; the case seemed so typical of fæcal absorption that it was decided to confine treatment to this factor alone. Colon douche was given daily; at first four pints, gradually increased until finally ten pints of warm water was used at each sitting, causing no pain or discomfort, showing the extremely atonic state of the large intestine. After twelve days the douche was given every second day, and later on twice a week.

The arthritis gradually and steadily improved. No other treatment whatever was given, and when last seen, June, 1899, the patient was quite well.

Dr. Garrod drew attention to the resemblance between the articular lesions of rheumatoid arthritis and those met with in locomotor ataxia and other nervous diseases, and Dr. Hawthorne made similar observations; but the latter commented upon the absence of pathological changes in the cord. On this point, it seems to me probable that while the more serious joint lesions, such as Charcot's, are due to organic lesions in the cord, the slighter changes, causing arthritis, are probably only functional.

The influence of influenza in causing acute arthritis has already been mentioned. It is also a factor of great importance in many of the slower cases.

Case 10. *Influenza; hard work and anxiety; arthritis.*—S. B—, female, æt. 35, hospital nurse. Never had gout or rheumatism. In 1895, while run down by heavy district nursing, contracted influenza; returned to work too soon; had great anxiety due to family troubles. Swelling of joints came on, fingers and shoulders at first, followed by knees, ankles, wrists, and elbows. Slight rise of night temperature from time to time. Some spasmodic contractures of elbows, wrists, and knees; gradually got worse till 1897; meanwhile made very anxious by the gradual absorption of her small savings.

October, 1897, came to Buxton. Much muscular atrophy and neuritis; very much crippled.

Had constant current electric baths, a generous dietary, port wine, and Liq. Auri et Arsen. Bromid.; 1898 and 1899, courses of electric baths; gradual but continuous improvement, but has still some swelling and contraction of joints, a certain amount of which will no doubt be permanent.

Dr. Hawthorne mentioned Sir James Paget's views on the production of atrophy in the neighbourhood of an affected joint, viz. that an irritation was carried by the afferent nerves of the joint to the cord, set up mischief there, and the result was

Digitized by Google

transmitted back to the structures near the affected joint, injuring their nutrition and vitality. I believe that it is this action which causes general arthritis to commence from one injured joint—the so-called traumatic group.

CASE 11. *Injury to knee; arthritis of both knee-joints; repeated effusion.*—E. C—, male æt. 35. "Not very well for twelve months;" then fell from cycle, injuring left knee. Gradually both knees became painful and swollen; repeated recurrent effusions into first one joint and then the other. Knees had been kept on back splints and painted with iodine for two months, but pain had not decreased. Then very hot douche of brine, which made joints worse.

June, 1898, came to Buxton. Gentle rubbing with ice night and morning for half an hour. Guaiacol carbonate given, knees still kept on splints. After ten days of the ice treatment gentle passive movement was given, and the ice massage reduced to once daily. The patient was kept for nine months on guaiacol carbonate, and cod-liver oil emulsion with hypophosphites.

November, 1899, "quite well;" no return of joint trouble.

I have a series of sixteen cases in which injury to the nerve below the deltoid has caused an ascending neuritis reaching the spinal cord, and afterwards followed by ankylosis of the shoulder.

CASE 12. *Injury to shoulder; acute ascending neuritis; spreading arthritis.*—V. T—, female æt. 50. January, 1896, injury to right shoulder, followed by acute neuritis, which extended up to the spine. Gradually left shoulder and both wrists became swollen and painful, and partial ankylosis of right shoulder came on.

October, 1896, came to Buxton much reduced in health and greatly depressed by pain and suffering, the "toothachy" pain preventing sleep. She drank the Buxton thermal water, had the combined vapour and mineral baths, and later on mineral and constant current electric baths. Steady improvement took place. June, 1897, much improved, and had a short bath course as before. June, 1899, "quite well."

The bacillus discovered by Drs. Bannatyne, Wohlmann, and Blaxall in rheumatoid joints has not as yet reproduced arthritis on injection, although a somewhat similar micro-organism is alleged to have done so in the hands of German investigators.

The bacterial origin of this disease does not seem as yet to have been proved, and although such a form may, and probably does constitute one of the varieties of rheumatoid arthritis, it is difficult to see how this factor could account for all the varied forms in which arthritis shows itself. My own experience inclines me strongly to the belief that under the name of rheumatoid arthritis are grouped together cases which, while they unite in showing more or less disturbance of the nerve nutrition of the affected joint or joints, differ in other important particulars. The base upon which the disease is built undoubtedly varies, and reflex irritation from various sources often seems to influence its inception and continuance.

Remak held that certain articular affections were due to irritative conditions of the spinal marrow and of the sympathetic nervous system.

Ord pointed out three important facts:

(1) That certain changes in the nutrition of the joints take place through the influence of the nervous system.

(2) That certain organs, especially the uterus and ovaries, have the power, through centripetal nervous influence, of producing considerable excitement in the spinal cord.

(3) That in cases of debility or anæmia the reflex irritability of the spinal cord is greatly increased.

Whatever the variety of the disease, the subjects of it will almost always be found to have had the power of resistance of their nervous systems weakened by anxiety or other similar cause, or the reflex irritability of the spinal centres increased by debility of anæmia. When loss of resisting power or increase of irritability in the joint centres, or both, are present, it is not wonderful that joints, already the seat of inflammatory lesions, should take on changes which may both alter the character of the ailment, and also call for variation of treatment.

In conclusion I would submit to you—(1) That if one case of arthritis can be cured by simply washing out a long-loaded colon, and thus removing a source of auto-intoxication; another by an exclusive dietary of red meat and hot water, which can only act by checking gastric, and more especially duodenal fermentation, and by stimulating the nervous system to more complete excretion; a third by a removal of some source of

Digitized by Google

utero-ovarian irritation; and if the most generally useful therapeutic means are those acting upon the spinal nervous system, it is certainly improbable that all rheumatoid arthritis is due to a bacterial factor. (2) That, taking into consideration the large amount of neuritis continually present in rheumatoid arthritis, the marked muscular atrophy and the general resemblance to joint affections known to proceed from disease of the spinal cord, it seems likely that the disease is a dystrophy dependent either upon central nerve disturbance or upon reflex irritation transmitted to the joint centres from some other organ. (3) That this irritation is very apt to be directed toward any joint already affected by rheumatism or gout. (4) That acute rheumatism is occasionally followed by a form of rheumatoid arthritis. (5) But that, so far as present knowledge goes, there is no common origin of acute infective rheumatism and of the more chronic joint conditions we have been discussing this evening.

The CHAIRMAN: I think we have had a good representation of the current views from different aspects of the question. Still it is not for me to doubt that from the body of this meeting some fresh light on the matter may arise. I think the meeting will join with me when I invite Dr. Paine to favour us with a few remarks, even though we have heard the joint paper of himself and Dr. Poynton.

Dr. ALEXANDER PAINE: I have very little to contribute to the discussion, because all I have had to say was embodied in the joint paper which was read by Dr. Poynton. But there is one observation I should like to make, and that is to correct an error in Dr. Armstrong's paper. He said he supposed that the rabbit which had the affected joint only lived eight days. As a matter of fact the rabbit lived from May to October, which is nearer eight months than eight days. We could scarcely regard an eight days' affection as chronic. As regards his remarks on the early symptoms, which I take it were those more or less of what we should understand as malaise, many cases which are exposed to damp he seems to regard as rheumatic. Well, if they are rheumatic in the sense of acute rheumatism, then it is quite consistent that such symptoms may be due not necessarily to the prevalence of the organism, but of the toxins of the organism. Of course one cannot speak definitely yet on the question of toxins with regard to this organism, because we know very little of the organism itself; it is not until we have isolated the toxins that we shall be able to pass any definite opinion and say what symptoms are actually due irritatively to the presence of the organism and what is due to the action of the toxins. But I reiterate it is possible that many of the symptoms that come on even before the sore throat, may be due to the absorption of toxins. We find that also in other diseases; in influenza, pain in the back and other disturbances are entirely due to the absorption of the toxins, because in that disease we do not find an organism, except in the very severest cases, entering the system either by the lymphatics or by the blood-stream. The part that cold and damp play in the causation of rheumatism as an infectious disease is not different from what it is in other infectious diseases,—that is to say, it acts probably by lowering the resistance. There was a time, which I can remember well, when acute lobar pneumonia, which we now know to be due to the pneumococcus of Fraenkel, was ascribed to cold and chill. Chill has been so wrapped up with rheumatism that it is rather difficult to get away from the idea that the disease itself is not in many of the early symptoms possibly due to cold alone. But Dr. Newsholme, in his statistics and in his Milroy Lectures, pointed out that rheumatism is not always associated with damp and cold, and he found that it occurred not on the clay soils but rather on the dry soils, and where there was a low ground water. That which he pointed out was therefore very much in favour of the infectious origin of the disease. I will not now trouble you by going into the other features of the disease which should confirm our view that it is infectious.

Mr. ARMSTRONG: I regret the error to which Dr. Paine alludes, but I had a full report of the previous discussion taken for me, and the error was made in that.

Dr. BLANC (Aix-les-Bains): I have been a long time among people who have been sufferers from rheumatism and rheumatoid arthritis, and I cannot help thinking that rheumatism is a distinct disease from rheumatoid arthritis. Some people are of opinion that rheumatoid arthritis arises from special toxins or from particular foods; but it seems to me it comes from other troubles which we do not

Digitized by Google

know of, because it is a disease which is of long standing, and it seems difficult to reconcile that with a toxic character. You may find rheumatoid arthritis persisting for one, two, three, four, or even five years. Then it may be quiescent for a year, and after that will return to activity. I am asked why have I rheumatism? Some say I have a pain here, others say I have a pain in my knee. But that is not acute rheumatism, it is a special kind of chronic rheumatism which has nothing to do with rheumatoid arthritis. It is certainly very often a consequence of true rheumatic fever, but very often the rheumatic fever is a different disease. You have to remember how the so-called rheumatoid arthritis patients are quickly cured. You will have noticed in your practice how often one man has benefit from a certain remedy which another man does not get. I would like to ask anyone here if he cures true rheumatoid arthritis.

Dr. Garrod, in replying on the discussion, said: I do not think there is much difference of opinion amongst us; that is to say, I think we all agree in thinking that the whole question is one which is still involved in very great difficulty, and that the subject which you have laid down for discussion is one which still admits of discussion, because we are none of us quite confident of what we mean when we use the terms chronic rheumatism, rheumatoid arthritis, and rheumatoid affections. Perhaps you will allow me to refer briefly to the remarks which have been made by the different speakers, and any other points which occurred to me when listening to the various most interesting and valuable remarks which have been made.

We are all familiar now with the bacteriological work which has been done in connection with acute rheumatism by Drs. Poynton and Paine, and we feel strong hope that further experience will teach them, and that other observers will confirm the same, that we have in the diplococcus which they have described actually arrived at the pathological cause of acute rheumatism. Naturally Drs. Poynton and Paine have devoted their attention chiefly to acute rheumatism, but I think their contribution to this debate has been of very great value to us all, because it has given us a criterion by which to differentiate acute rheumatism from other affections; that is to say, when we speak of rheumatism we mean a definite disease, and a disease which in all probability is caused by a specific bacillus.

Mr. Macnamara's remarks were, I think, of great interest, and I think they are of great value, because they will lead us all to carefully look for all possible sources of infection, such as diseased condition of the mouth, which, I take it, Mr. Macnamara regards as only one of the possible sources of infection, or starting-points of disease, or some of the diseases known as rheumatoid arthritis.

Dr. Hawthorne's remarks were also, I think, of extreme value, and I rather regret that the view which formerly certainly was widely held, and which is associated with such honoured names as those of Charcot and Pasteur and others, has not found more advocates in the course of our debate, because I think it would have given rise to more active discussion. Perhaps the most controversial point which has been raised in this discussion has been as to whether or not rheumatoid arthritis so called is merely a continuation of an actual rheumatic affection. It is a curious fact—as showing the alteration in the drift of opinion—that in former discussions which have taken place at various associations in this country and in America in recent years the bulk of the opinion has rather been contrary to that expressed in our debate during these two evenings. At a British Medical Association meeting a few years ago, if I remember aright, and also, I think, at the American Medical Association, the bulk of the opinion was rather in favour of the rheumatic nature of these affections; and quite recently there have been papers written setting forth that view somewhat strongly. I take it that Dr. Hawthorne did not appear as a very enthusiastic advocate of any one view. He rather took a judicial standpoint, and urged that we must not dismiss it as worthless because it happens not to be the prevalent opinion at the present moment. His arguments in favour of further consideration I think demand great attention. It was with extreme interest that I listened to Dr. Hewlett's remarks, particularly on the bacteriological questions involved, and his estimates of the value of the various bacteriological researches which have been carried out on both diseases.

Mr. Arbuthnot Lane spoke on an aspect of the question which has been too much neglected, and I think I understood him to say that the question of traumatism which he dwelt upon related

Digitized by Google

chiefly to the more chronic single joint affections of elderly people, and that he himself differentiates quite distinctly the more acute forms of multiple rheumatoid arthritis as quite a distinct thing, and as not having very much to do with traumatism.

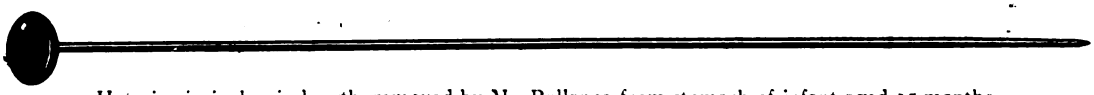
With what Dr. Bannatyne said I find myself very much in agreement. It seems to me that he takes views very similar to those which I endeavoured to set forth in opening this discussion, namely, that acute rheumatism and what we call rheumatoid arthritis have no very definite connection, and that under the name rheumatoid arthritis we are speaking not of one single affection, but of a group of affections which are required to be carefully distinguished from each other. I do not think there is any difference between us on that point. Dr. Armstrong's remarks also seemed to possess a special interest on account of the stress which he laid on the very various factors which come in in the ætiology of the various cases. He dwelt on an aspect of the question which I think other speakers have touched upon but very little,—that is to say, how in one case one seems to trace one definite factor, and in another case some factor of quite a different nature. The work of the future will be to reconcile these, to see whether they are all as distinct as they appear at first sight, and whether they may not all produce their effects alone in a somewhat similar way,—that is to say, whether there may not be one common active factor which is caused by various exciting causes. I can only conclude, sir, by saying that I think this discussion ought to do a great deal to clear the ground, and to say once more that I think we are only beginning to learn something about the conditions grouped together under the name of rheumatoid arthritis. There is a preliminary clearing of the ground, and now that we have got rid of the trees to some extent, it is our duty to begin to build.

WITH MR. BALLANCE AT ST. THOMAS'S HOSPITAL.

Removal of a Hat Pin Six Inches Long from the Stomach of an Infant Fifteen Months Old.

S. W—, male, æt. 15 months, was admitted on March 10th, 1901. Eighteen hours before admission, whilst standing upon a chair, the child placed a lady's hat pin (see fig.) head foremost in his mouth. He fell from the chair, and the pin, striking the ground, was pushed down his throat. His mother, seeing the sharp point sticking upwards, tried to pull it out, but succeeded in pushing it further down. The child subsequently swallowed two pieces of toast without any difficulty. When brought up to the hospital, the child exhibited no signs of anything wrong. Two skiagrams were taken which showed the pin to be in the lower end of the œsophagus, the head being about the level of the cardiac orifice of the stomach.

At the operation, which was performed thirty hours after the accident, the œsophagus was first explored with the finger, but nothing could be felt. An incision two and a half inches long was then made in the middle line between the xiphoid cartilage and the umbilicus. The stomach was exposed, and held up to the surface by two threads passed into its wall. A small incision was then made between the threads. The finger in the stomach found the head of the pin presenting at the cardiac opening, and it was extracted without difficulty. The wound in the stomach was closed with two rows of sutures and the abdominal wound was closed in layers. The pin was six inches in length. The temperature rose to 104° on the third day, but soon became normal, and convalescence was practically uninterrupted. For the first twenty-four hours the child was fed *per rectum*. On the second day one ounce of milk and barley water was given hourly, while on the fifth day he had bread and milk. He was discharged well on March 26th.



Hat pin six inches in length, removed by Mr. Ballance from stomach of infant aged 15 months.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 447. WEDNESDAY, MAY 22, 1901. Vol. XVIII. No. 5.

CONTENTS.

PAGE

*A Clinical Lecture on the Diagnosis of an Ovarian Cyst. By AMAND ROUTH, M.D., B.S., F.R.C.P. 65

*A Clinical Lecture on Some Cases of Tabes Dorsalis. By W. ALDREN TURNER, M.D., F.R.C.P. 71

*A Case of Multiple Subcutaneous Sarcomatous Tumour. Demonstrated by JONATHAN HUTCHINSON, F.R.C.S., F.R.S. 74

*The Practice of Blood-letting. By J. F. BRISCOE, M.R.C.S. 76

Creosotal (von Heyden) in Severe Scrofulous Affections and Ozæna ... 80

*Specially reported for the Clinical Journal. Revised by the Author.

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

THE DIAGNOSIS OF AN OVARIAN CYST.

A Clinical Lecture delivered at Charing Cross Hospital, March 5th, 1901,

By AMAND ROUTH, M.D., B.S., F.R.C.P.

GENTLEMEN,—I desire first of all to draw your attention to a diagram (Fig. 1), which represents the relations of the different pelvic organs in the female.

I want to go over some of the evidence which we may have as regards an abdominal tumour being an ovarian cyst. Here we have a woman with an abdominal tumour. The question is what is the exact nature of it. Yesterday we made a cursory examination and we thought that in all probability it is an ovarian cyst, and I want to go over some of the grounds upon which we formed that opinion. I have explained to you by the aid of the diagram what is the origin of ovarian cysts. They are broadly called "ovarian cysts," but incorrectly so, because you know that many of them, such as paroyarian cysts, do not develop from the ovary at all. The varieties of "ovarian cysts," speaking broadly, are nine or ten in number. First of all we have those derived from the oöphoron, that is the free part of the ovary, that part which bulges furthest from the back of the broad ligament. The oöphoron contains the matured Graafian follicles, and also corpora lutea, in various stages of development and retrogression. The ovarian tumours which spring from the oöphoron are cystic or solid. There are the single cysts developed from Graafian follicles, which are generally lined, therefore, by columnar epithelium, practically the remains of the tunica granulosa. The epithelial cells may be flattened by pressure and become stratified, or may entirely atrophy. Then there are multilocular ovarian cysts, which come from the distension of more than one Graafian follicle. Then there are the multilocular adenomatous cysts. In addition to these there are the dermoids. The difference between ordinary multilocular cysts, adenomatous cysts, and dermoids,

Digitized by Google

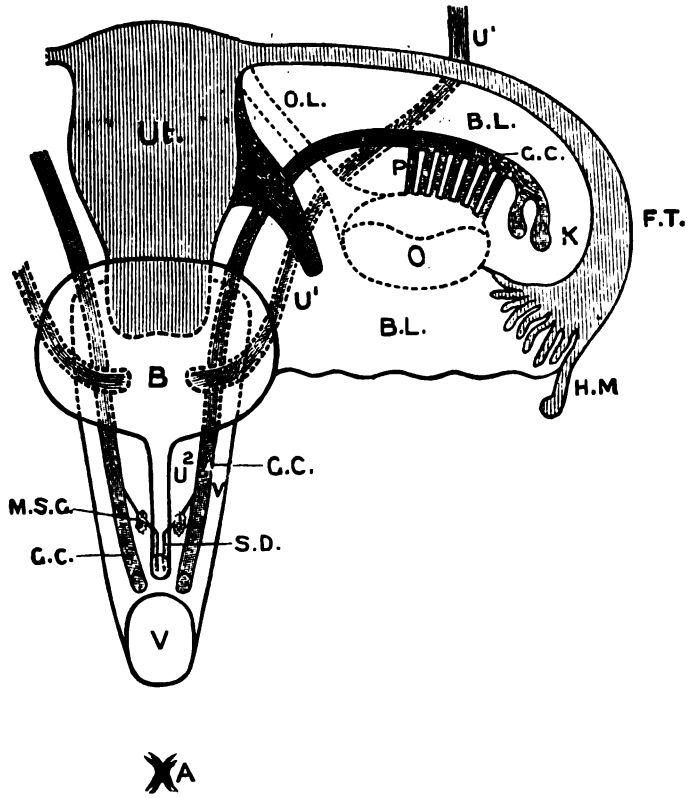


Fig. 1.—Ut. Uterus. B. Bladder. U^1. Ureter. U^2. Urethra. S.D. Skene's ducts. V. Vagina. G.C. Gartner's canals. M.S.G. Max Schüller's glands. K. Kobelt's tubules. A. Anus. B. L. Broad ligament. O. Ovary. O.L. Ovarian ligament. R.L. Round ligament. F.T. Fallopian tube. H.M. Hydatid of Morgagni. P. Vertical tubes of parovarium. (Modified from Skene.)

We are indebted to the Obstetrical Society for the loan of this block from Dr. A. Routh's paper on "Parovarian and Vaginal Cysts," vol. xxxvi, p. 161.

is really very slight. You have only to get the stratified and columnar epithelium converted into a structure where there are glands from which retention-cysts develop, and the cyst becomes adenomatous; and you have only to get a little hair or bone in connection with the skin or mucous membrane for it to be dermoid. There are certain solid tumours springing from the oöphoron—and we are still dealing with the oöphoron—and those are the fibromata, myomata, sarcomata, and, according to most, carcinomata, springing from the epithelium lining these different cysts. Cysts developed from the hilum of the ovary, or as it is called paroöphoron, have certain peculiarities. The Graafian follicles are immature, and very often indeed the cysts are lined by a fibrous tunic with modified epithelium, and they often have papillomatous growths on the inner surface, and are serious for this reason. Then we have the parovarian cysts, most of which spring from the vertical tubules. Seven of these tubules are depicted in Fig. 2, but sometimes

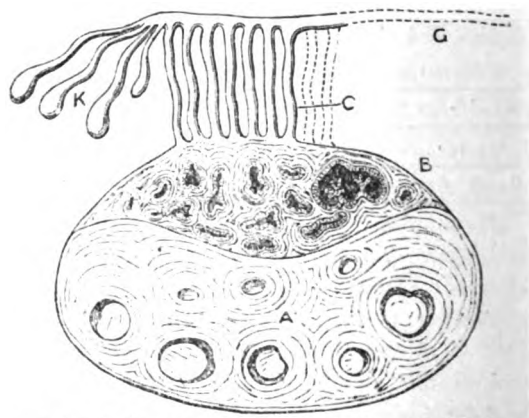


Fig. 2 (semi-diagrammatic).—A. Oöphoron. B. Paraoöphoron. K. Kobelt's tubes. C. Vertical tubes of the parovarium. G. Gartner's duct.

Digitized by Google

there are as many as twenty. These cysts develop more within the mesosalpinx or broad ligament than the hilum or paroöphoric cysts, which also burrow, but are not so definitely between the folds. In addition there are Gärtner's cysts, which develop in the longitudinal tube of the parovarium. As you will see from the diagram these cysts of the longitudinal tube may cause a distension of the whole tube right into the vagina; and in the case that this diagram was drawn to illustrate there was a large broad-ligament cyst, communicating with a vaginal cyst, which was lying along the roof of the vagina; there was vaginal impulse on coughing, and pressure on the vaginal cyst emptied its contents into the broad-ligament cyst. When I opened the vaginal part it drained the broad-ligament cyst and cured it. There are only one or two such cases on record, but this was a very definite one. The contents of these cysts, from a diagnostic point of view, are very unimportant. When cysts were treated by tapping, a good deal used to be made out of the examination of the contents, which were said to be almost diagnostic. But we know now that that is not so. The other day we got some help in this respect in No. 9 bed, from examining under the microscope the fluid contents of an abdomen, and the condition turned out to be, as we had diagnosed, an old ruptured papillomatous cyst. Sometimes in these ovarian cysts one finds columnar epithelium, sometimes large granular cells, such as Drysdale or Gluge cells; but these latter are not necessarily diagnostic of ovarian cysts.

Now the relations of ovarian cysts differ according to their position at the time. For instance, taking a cyst of the oöphoron when it is small, it bulges out from the back of the broad ligament. As it gets heavier, as it tends to do, it drags down the broad ligament, and by gravity gets into Douglas's pouch. The immediate effect of that is to necessarily displace the uterus, either to the other side—if it is at first in one of the posterior quarters of the pelvis, or forwards, against the pubes, if the cyst is in Douglas's pouch. You will see represented in the diagram the ovarian ligament, which is perhaps one inch or one and a quarter inches long, and it acts in such a way that the ovary, instead of going straight down, describes a half circle with the uterine cornu as a centre, and gets first into one or other posterior quarter of the pelvis and then behind the uterus. The result is that when one examines the case *per vaginam* the ovary, if small, is found in a definite pelvic relation to the uterus. When it gets larger it tends to rise out of the pelvis. If it is in Douglas's pouch below the sacral prominence, it goes on enlarging until it is inconvenient to remain there, and then its upper surface enlarges until it rises above the brim and draws up the pelvic part of the cyst, and finally comes to rest on the pelvic brim all round.

The prognosis in the case of an ovarian cyst is always bad if it is not dealt with surgically. If there be no operation the course is downhill. The patient gets worse and worse, and then come well-marked local symptoms from pressure in the pelvis, on the bladder and uterus, and on veins, ureters, and on the sciatic nerves. Eventually the tumour rises out of the pelvis, and for a time much relief is experienced. Afterwards other structures are pressed upon, such as the vena cava, giving rise to large veins over the abdomen; or the bowel with resulting intestinal dyspepsia; or when the cyst is large it may press on the stomach, or even on the diaphragm, causing dyspepsia and dyspnœa; or on the ureters or renal vessels, leading to trouble in the kidneys. Thus the main symptoms of ovarian cysts are those produced by pressure and by the interference with nutrition consequent upon disordered function.

We must not forget in dealing with ovarian cysts that, putting aside any pressure results, there is a very strong tendency to complications. In the middle stage, when a cyst is of the size of a fist or a cocoa-nut, it is apt to get a twisted pedicle. When this occurs the arteries are still able to pump blood into the ovary continually, but the blood cannot return, owing to the veins becoming blocked. The result of that is that some vein on the inner surface gives way, hæmorrhage into the cyst takes place, and rupture may follow. Apart from that, however, inflammation may occur with adhesions to other organs, and the cyst may even suppurate and open into some viscus, and septicæmia may ensue.

Well, now, here is a patient with an abdominal tumour, and it has to be distinguished from a good many things. I do not want to take the various conditions which it may be, and disprove them in

Digitized by Google

separate order, but we will go through the physical signs as one finds them, pointing out the differences which would be found if the tumour were not an ovarian cyst.

Amongst the more ordinary abdominal swellings are pregnancy, a full bladder, and a fibroid of the uterus, and, perhaps I should add, ascites. In addition there are renal tumours, splenic tumours, hepatic tumours, gall-bladder tumours, omental tumours, and the more fixed tumours, such as adhesive peritonitis, encysted serous peritonitis, tubercular peritonitis, or malignant growths.

When one first sees a patient it is useful to try to get some sort of history. The history, therefore, is what one has to take first. Now pregnancy would have a definite history of its own if the patient could be relied upon. Here we have a three months' amenorrhœa, but the patient is forty-seven years of age. At the age of forty-seven a three months' amenorrhœa is not unusual, owing to the advent of the menopause, and it would be very unlikely that a woman aged forty-seven would be pregnant. You can see, also, that in this case a three months' amenorrhœa does not correspond with a tumour rising above the navel to the size of an eight months' pregnancy. The patient has not been ill, but has enjoyed very fair health all along; whereas, if this had been ascites she would have been ill with it in some way, for whether the cause be a general one, such as diseases of the heart, or liver, or kidneys, or due to a local tumour, the patient would be manifestly ill.

In the present case the patient only recently realised that she was getting large in the abdomen, and until three or four weeks ago she does not seem to have suffered from any marked constitutional or local trouble. So from the history one has to think of something which is not pregnancy, and is slowly and steadily increasing without giving rise to any very serious disturbances. A catheter has excluded the possibility of a distended bladder. The amenorrhœa at forty-seven years of age is against the tumour being a fibroid of the uterus. As far as physical signs are concerned, those who are opposite to the patient will notice that the abdomen is a little bit irregular in shape. It bulges a little on the right side, and is a little flat on the opposite side, and it bulges at the left lower part. A cyst of this size, reaching a hand's breadth above the navel, generally presents those characters; it appears to be pretty central, but there is a bulge in one or other direction. If the cyst were seen in an earlier stage, say when it was half or one-third the size of the present one, one would find it occupying a more or less lateral position; that is to say, if it sprang from the right ovary, its greatest half would tend to be on the right side of the belly.

Further, by inspection one can see a few veins marked over the surface of the abdomen. There are a few lineæ albicantes, mostly of old standing, though there are also a few recent ones. It appears she had her last child ten years ago. In ovarian dropsy, as a rule, there is not much bulging at the flanks, but here there is a little more than one generally sees. But you see the cyst is of good size, reaching almost to the ensiform cartilage, so that a good deal of room is wanted for it, and the bowels are pushed down partly into the flanks. Then again, by prolonged watching one can see no alteration in shape, as there would be if it were a pregnant uterus, for, during an intermittent contraction the tumour is thrown forwards and the flanks are less distended. That is because the uterus hardens all over, and instead of resting on the spinal column and bulging a little on each side when relaxed, it gets rigid and lengthens, and is thrown forwards against the abdominal wall. We have not noticed that here.

Palpation is of great value here. On passing one's hand over the tumour one finds there is no such thing as any very local nodule. There are large bulges everywhere in different places, but there is no nodule such as one would get in a fibroid; sometimes in fibroids the nodules are quite movable over the surface of the tumour; they become subperitoneal and get a little pedunculated, and one can move them freely. That is diagnostic of a fibroid tumour of the uterus. Here there is nothing of the sort; there is simply bulging. With regard to the consistence of this tumour, it is uniformly elastic, not hard at one point and soft at another. In most ovarians that is the case, because the solid portions of an ovarian tumour are, as a rule, on the pelvic aspect of it. In ovarians generally the cyst itself, or the series of cysts, bulges out upwards. Occasionally one does feel solid lumps in connection with ovarian tumours, and this greatly increases the difficulty of

Digitized by Google

distinguishing such a cystic tumour from a fibroid. Here there is nothing of that sort to complicate matters. Then we cannot detect by the hand, any more than we could by inspection, anything in the nature of contractions, which fact points to its being not any muscular tumour, such as a pregnant uterus.

The next point is as regards fluctuation. In a unilocular ovarian cyst one can almost always get a wave of fluid the moment one taps it. If one puts one's flat hand on one side of the abdomen, and flips with the finger on the other side, a definite wave can be detected. That is the case also in ascites. Fluctuation proves a collection of fluid, in the one case encysted, and in the other free in the abdominal cavity. Here it is not possible to get a distinct wave except over small areas, but the tumour is extremely elastic in all directions over the whole surface. The probability is that in this case we have to deal not with a unilocular cyst containing thin fluid, but with a multilocular cyst, and I think it will be found to be a cyst with several very large cavities, probably each of them filled with that very viscid secretion which is found in adenomatous cysts. The bulgings also in this tumour point to the cyst not being unilocular. In this case there is an entire absence of any such sensations as would be produced by a fœtus moving freely in liquor amnii, such as fœtal movements, fœtal outline, or abdominal ballottement. The percussion note over a cyst, over a pregnant uterus, and over a fibroid is practically the same, that is to say it is dull all over, wherever you percuss it, and it becomes resonant in all directions towards its edges. It is resonant because a tumour like this growing from the pelvis gradually pushes the small intestine in front of it, and also to some extent the colon and the stomach. Supposing it to be an ovarian cyst, one ought to find that it is resonant as one gets into the epigastrium, and in each flank one can palpate the cyst at a level higher than the dulness extends to. That is because as the cyst rounds off there is room for a piece of small intestine to nestle just where it is passing away from the abdominal wall, and the result is that it becomes resonant there. Here I can feel the tumour another finger's breadth above the line of limit of dulness. In the flanks one gets a definite colon note.

In cases of ascites the percussion notes would be almost exactly opposite. The water, in obedience to its hydrostatic properties, would gravitate down and lay at a perfectly even level, and the intestines, containing air, would float above it, and give you a resonant note above the level of fluid. The dull areas and clear areas vary in ascites according to the position of the patient. As this patient turns over, we find the tumour is still absolutely dull until we come to the resonance of the colon in the loin. The relative areas of dulness and resonance are unaltered.

Every now and then those signs mislead us. For instance, one may find the colon so full of fæcal matter and very little gas, that it will give a dull note down in the flank. Or one may find dulness in the flanks in the case of an ovarian tumour complicated by the presence of free fluid. In some cases of ascitic fluid one may find the abdomen clear and resonant in the flanks, due to the colon being adherent.

Then with regard to auscultation. If you listen over an abdominal tumour you may find a good many signs by means of the stethoscope. When it is a pregnant uterus, especially if the tumour be of this size, you will probably hear the fœtal heart, and in the majority of cases it is heard to the left and below the umbilicus, owing to the first vertex position being so frequent. You may also sometimes hear other fœtal signs, due to movements. If the uterus is twisted and oblique to the right, as it often is, you will get the left broad ligament brought to the front, and you will hear a uterine souffle over the vessels of the left broad ligament, synchronous with the mother's pulse. In cases of fibroid you will often hear this uterine souffle of a harsher type than the souffle of pregnancy. Such souffles are very seldom noticed in the case of ovarian tumours. Yesterday we found that this particular tumour is dumb, no sounds whatever being heard. Although one could detect the thud of the abdominal aorta, as conveyed through the tumour, there is no blowing or other sound to be heard.

The points which we have gone over serve to distinguish this condition from nearly every other form of tumour. We have distinguished it from pregnancy by the fact that there are not fœtal outlines or movements, there are no evidences of a contracting uterus, and there are no other auscultatory signs. It is distinguished from ascites

Digitized by Google

because of its permanent areas of dulness and resonance.

But there are certain other tumours which such a swelling as this may possibly be; for instance, a renal tumour. But in that case it would be more or less one-sided, and the tumour would, on one side or other, be traceable down into the kidney region, and the colon would give a clear note across the tumour. If it was a splenic tumour it would be dull to the left ribs. Moreover, the tumour is of a different shape to what it would be if connected with the spleen. It is not flattened anywhere, but is rounded all over, and no dent can be felt. One is aware that in operations for what is believed to be ovarian cyst one now and then comes across a case where one is surprised to find something else. I think the commonest of these unlooked-for conditions is a hydronephrotic kidney. However, there is no suspicion of that here.

On coming to examine this case *per vaginam* we find that the uterus is at a low level, and that apparently it is a small uterus. I tried yesterday to pass the sound, but failed, because she was lying on the bed, and the cervix was so movable that I could not fix it sufficiently to pass the sound in. As soon as the point of the sound reached the internal os, the cervix slipped away. As far as we could find, the uterus is a little retroverted, depressed by this large ovarian cyst. The uterus is very often in that position in these circumstances. As the ovarian tumour becomes large enough to get out of the sacral hollow it rises up into the abdomen, and may lie upon an anteverted uterus. As a rule, it gets in front of the uterus, which is retroverted, with a tendency to later version owing to traction on one corner. As a rule the sound passes the normal distance, two and a half inches. In this case, when I fix the cervix with a volsellum and draw it down, the sound passes about three inches. Having got the sound in, I can move the uterus away from the tumour. I can tilt the sound round, and with my finger in the vagina or rectum, I can feel the end of the sound, especially when I thus draw the uterus. I am thus able to make sure that the uterus and the ovarian cyst are quite distinct, that one freely moves over the other, with no closer attachment than from a pedicle which has become much stretched. By removing the volsellum I can elevate the uterus by the sound, and feel the fundus by my abdominal hand depressed between the cyst and the pubes. These points and the history and the other physical signs, prove it is not a fibroid tumour of the uterus.

Sometimes the uterus may be found to be adherent to the front of the ovarian cyst. As the cyst grows the uterus is stretched, so that occasionally one finds an ovarian cyst with the uterine sound passing five inches or more, and going apparently into part of the cyst or tumour. It is very difficult to be quite sure in these cases that you have not to do with a fibroid tumour in one wall of the uterus, the cavity of the uterus with a thinned opposite wall being in front of it. The distinction is sometimes made in these cases by the fact that, when the uterus is drawn up by a cyst, you can feel the two-fingers'-breadth uterus lying in front of the cyst above the pubes, and the sound goes up into it. Sometimes bimanually you can dip your hand down below the cyst and feel the fundus. I could not quite do so here, till I had elevated the uterus on the sound as above stated.

These are the main points which enable one to feel pretty clear that this is an ovarian cyst.

The Pupil after Death.—J. E. Courtney, in the 'Medical Record' of March 2nd, 1901, says that ordinarily the pupils are quite widely dilated and about equal after death, and shortly become fixed in a position of moderate dilatation. This is true in a majority of cases, but a considerable number of autopsies in which the condition of the pupil is observed will show exceptions. His observations convince him that in certain cases of disease of the brain the pupils may retain after death the impress of the condition imparted to them by the disease which caused dissolution. If this fact should be established it might be of service in suggesting the cause of dissolution in persons found dead. There appears nothing inconsistent in the idea of the muscular fibres of the iris becoming fixed in about the position in which they were at the time of death. In one case, in which the patient died of a cerebral hæmorrhage, the pupils were dilated on the side opposite the lesion, and remained perceptibly so at the time of autopsy, ten hours after death.—*Medicine.*

Digitized by Google

A CLINICAL LECTURE

ON

SOME CASES OF TABES DORSALIS.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London.

By W. ALDREN TURNER, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

GENTLEMEN,—I propose to-day to show you a series of cases of locomotor ataxy, several interesting examples of which are present in the Hospital. The cases show not so much the different stages of the disease which are described in the text-books, as the complications and variations which are met in practice. But a few preliminary remarks may be made upon the three stages through which the disease is said to pass.

The first, also called the pre-ataxic stage, is chiefly characterised by the presence of lightning pains, and by the absence of ataxy or incoordination of the voluntary movements. But pains, sharp and shooting, are by no means the only feature of this stage. Various other phenomena may, indeed, give the clue to the true nature of the pain, which the patient most probably has ascribed to "rheumatism." Of these may be mentioned perforating ulcer on the sole of the foot, in many cases one of the earliest symptoms of tabes dorsalis; severe attacks of vomiting and pain in the belly, lasting a day or two or even longer, producing great exhaustion, and sometimes collapse, and paroxysmal in character:—gastric crises; difficulty sometimes in passing urine, at other times in retaining it, the latter being most commonly of the nature of overflow from a full bladder; and diminution or even loss of sexual desire and power.

The existence of one or more of these phenomena should draw the physician's attention to the state of two important reflexes, viz. the knee-jerks, and the pupillary light reaction. In the majority of cases of early tabes, one or both of these reflex actions will have disappeared.

Case 1 illustrates in a typical way, the early stage of tabes.

1. A. B—, æt. 38, had a hard chancre fourteen years ago. For six or seven years he has suffered from pains in the limbs, more severe in the legs than in the arms. He believed they were of rheumatic nature, as they were usually relieved by salicylate of soda. His attention was early attracted to the sole of the right foot, where, upon the prominence at the base of the big toe a troublesome "corn" existed. The co-existence of this corn with lightning pains naturally called attention to the condition of the knee-jerks and the pupils; the former were defective, being only brought out upon reinforcement, while the latter were small and presented a very feeble light reaction. There was no bladder trouble, nor any sign of ataxy, even when put to a severe test, such as walking along a straight line.

The following series of cases illustrate the character and variations of the so-called second stage.*

2. *Tabes with palsy of cranial nerves.*—This is the case of A. S—, æt. 40. He had syphilis five years ago. He has been ill twenty months, and the disease commenced in the common way, with some loss of control over the bladder, numbness, and later on shooting pains in the legs, and some difficulty in walking. I show him to you specially for this reason, that in addition to the ordinary symptoms of locomotor ataxy he presents paralysis of several of the cranial nerves. He shows a slight degree of proptosis, and some bilateral paralysis of the soft palate. Laryngoscopic examination reveals the presence of both adductor and abductor paralysis of the vocal cords. The tongue is protruded well, but with a certain amount of tremor. Although there is paralysis of the vocal cords, and paresis of the soft palate, there is no paralysis of the tongue or the lips, facts showing that the lesion involves most probably what is known as the nucleus ambiguus, that is, the nucleus of the motor fibres of the vago-glossopharyngeal nerve. This is a condition which is also found in syringomyelia, when neuroglial overgrowth implicates this part of the central nervous system. Of the general symptoms indicating the existence of tabes are: a small pupil, which is inactive to light; and absence of the deep reflexes, such as the knee-jerk, which cannot be elicited even on reinforcement. He has gastric crises, and has had them since the

* For the notes of the cases, I am indebted to Dr. Edwin Bramwell, late house physician; and for permission to refer to them to the physicians of the hospital.

Digitized by Google

commencement of his illness. Gastric crises, though commonly an early symptom, may exist throughout the disease. There is great ataxy in the legs, and you will notice that the right leg especially is thrown out in a very characteristic way. This is a typical case of tabes dorsalis characterised chiefly by ataxia, but associated with paralysis of some of the bulbar nerves, chiefly those supplying the vocal cords and the soft palate. He has only been ill twenty months, and the disease commenced five years after syphilitic infection.

3. *An ordinary tabetic.*—The next case is that of a man, forty-three years of age, who has been ill ten months. The onset was similar to that in the previous case. He has some difficulty in passing his water, and a considerable degree of ataxy of the legs. Shortly after the onset of the symptoms, he found he was unable to stand or walk. He also presents, to some extent, double ptosis. But in this case the ptosis is incomplete, and the notes show that there has been some recovery from it. If asked to use his arms, as in touching the tip of the nose with the forefinger, there are seen "marked wandering movements" of the left hand when held out and when the eyes are closed. There is some cystitis. The deep reflexes are absent. There is immobility of the pupil to light, incontinence of urine, lightning pains, and the ataxy is so pronounced that he is unable to stand. He admits having had gonorrhœa but denies a venereal sore. There is, however, very marked pigmentation of the right shin, which suggests a syphilitic condition.

4. *Tabes with marked ataxy of the arms.*—The next is a case of locomotor ataxy occurring in an older man. He is fifty-four years of age. He had syphilis when he was twenty-eight. Eight years ago he began to notice numbness in the right little finger and the thigh, and the numbness gradually spread over the body. Two years later, *i. e.* six years ago, he had shooting pains and retention of urine, and also unsteadiness in walking. I show you this patient to illustrate, more especially, the ataxy of the arms. In addition to the syphilitic history and the degeneration which results from such infection, there is a considerable amount of vascular degeneration. There is no albumen in the urine, but a well-marked double *arcus senilis* is observed. His pupils are small and immobile. The deep reflexes are absent. The gait is ataxic, and the Romberg symptom is pronounced.

5. *Tabes with ulnar anæsthesia.*—I now show you a case illustrating anæsthesia along the course of the ulnar nerve, a condition which is frequently seen in locomotor ataxy. The man states that he has never been exposed to venereal disease of any sort. He is forty-four years of age and has been ill four years. At that time he complained of shooting pains in the legs. Then he had gastric crises, which lasted to an intense degree for eighteen months. The case in other respects resembles an ordinary case of locomotor ataxy. The pupils do not react to light, the gait is ataxic, and the Romberg symptom is well marked. There is difficulty in micturition; the knee-jerks are absent. He presents diminished sensibility to painful impressions (analgesia) over the course of the ulnar nerve, or more correctly along the ulnar side of the arm and forearm. It is in reality more extensive, and is not limited to the distribution of the ulnar nerve.

This is a case in which venereal infection is denied, nor is there any evidence of specific skin disease or cicatrix upon the body.

6. *Tabes with rectal and bladder symptoms and impotence.*—I now present to you a man æt. 42, who had syphilitic disease twelve years ago. He was treated for that for a fortnight. Two years ago he developed numbness about the shoulders and a frequent desire to go to stool. He had incontinence of urine and loss of both sexual desire and sexual power. He also had double vision and unsteadiness in the movements of his legs. He has had pains in the knees, but not the characteristic lightning pains. He cannot walk by himself, and when assisted only does so with very great difficulty. He has no sense of possession of his legs at all. There is almost an absence of ataxy in the arms. So that this is a case of tabes chiefly affecting the lower half of the spinal cord, that is to say, affecting chiefly the lumbar region and the lumbar centres. The notes do not say whether there is paralysis of the sphincter ani in this case, though it is probable; that is to say, the sphincter ani does not grip the finger when it is inserted into the rectum. The loss of sexual power and sexual desire calls one's attention to the general question of impotence. I think we may say we have to deal with three kinds of impotence, or, rather, we have to deal with impotence

Digitized by Google

from three different causes. The first, and probably the rarest, is a congenital condition, due either to non-descent of the testicles, or to atrophy of the testicles, or to obliteration of the spermatic cord. In these cases the penis is well developed, but on squeezing the testicles there will probably be no "testicular" sensation, nor yet when the vas deferens is rolled between the finger and thumb. You will also find the vas deferens much shrunken in size in these cases. In all cases it is desirable, especially in young men, to make an examination of the actual organ of generation, to see whether there is some congenital defect. The next cause, and probably by far the most frequent, is neurasthenia. There is little likelihood of a history of syphilis in these cases. That is to say, if a person complains of impotence and denies venereal infection, the symptom is probably neurasthenic in origin, rather than from organic spinal disease. But if impotence is associated with paralysis of the bladder and rectum, it is of tabetic nature, and there exist other symptoms indicating some affection of the lumbar region. In tabes you will find the knee-jerks have materially diminished or disappeared, whereas in neurasthenia the knee-jerks will be active and will remain so. I think one cannot be too careful in giving a prognosis in cases of impotence.

7. *Tabes with Charcot's disease.*—The next patient is one about whom I have not got the history, but I show him to you because he presents an early Charcot's affection of the left ankle. He had an injury to the ankle three months ago. A skiagram has not yet been taken of it. You will notice a certain amount of swelling in the joint, but there is yet very little crepitation, though there is some roughness. It is quite painless. He has not done any work since last September, as he has not been able to walk since then. He does not have many pains. The case illustrates how an injury may give rise to or intensify an arthritic condition in tabes, which ultimately becomes a feature of the disease.

8. *Female tabetic with Charcot's disease.*—There is upstairs a female tabetic who is unable to stand, owing to dislocation of the hip taking place when she attempts to do so. I show you a skiagram of her left hip-joint, showing a characteristic condition. The skiagram is taken from behind. The neck and head of the femur have entirely disappeared, and palpation of the pelvis from the front reveals a distinct swelling in the left iliac region.

The *second* stage of tabes dorsalis is therefore characterised chiefly by ataxy, but, as we have seen, may be associated with a number of phenomena appearing as variations and complications, but in reality highly characteristic of the disease. Of these we have demonstrated paralysis of the cranial nerves, ulnar anæsthesia, sexual impotence, paralysis of the bladder and rectum, and Charcot's joint affection.

The third stage is merely an exaggerated continuation of the second, in which, owing to the intensity of the ataxy, the condition of the joints, or various intercurrent affections, the patient becoming bedridden.

Treatment.—In the treatment of tabes there are two chief factors to be borne in mind: first, the effort to combat the morbid process at work, an effort which is only likely to be of value in the early months of the disease; and secondly, the handling of its most pronounced symptom—ataxy. Bearing in mind the syphilitic causation of the disease in the majority of cases, the question arises whether a course of anti-syphilitic treatment is likely to be of any avail. Erb's view is that all such cases coming under treatment for the first time in the early months of the disease, should be given the benefit of this treatment. My own experience has been, that after the onset of ataxy, little good is to be obtained from the antisyphilitic methods. The cases which would seem to benefit most by a thorough course of mercury and iodide are those in whom the interval between onset of the symptoms and the syphilitic infection is short, that is to say, up to about five years; those in whom there is other evidence of the active working of the syphilitic poison, and those cases of early tabes characterised especially by pain in the back and tenderness on percussion of the spine, a symptom which would seem to point especially to a subacute or subchronic spinal meningitis. If antisyphilitic treatment is determined upon, it should be conducted upon the usual lines for six weeks or two months, and then recourse had to ordinary tonic treatment and rest. A further spell of mercurial inunctions may be advisable after a few months in some cases.

Fraenkel's exercises.—In an average case of tabes in the second stage ataxy and inco-ordination of

Digitized by Google

movement is the most obtrusive and trying symptom. It is now clear that much may be done to relieve it, the counteracting measures consisting of a series of graduated and systematic exercises, the patient learning in this way to overcome the ataxy by the carrying out of simple and systematic movements.

The exercises have been chiefly elaborated by Dr. Fraenkel of Heiden, and from this fact his name has been associated with them. In character they are simple, and are of such a nature that skill rather than force is required. Being devised to re-educate the movements of the ataxic, the patient's attention should be concentrated upon the work before him. The extent of the ataxy does not necessarily militate against their use, although it is obvious that a minor degree is naturally more favourable than if it is considerable.

In many cases, owing to the disturbance of the equilibrium, the movement may be carried out in bed. Lying on his back, the patient may be put through the common movements at the several joints of the legs, flexion, extension, adduction, rotation, or a simple apparatus, having a number of cup-shaped depressions along one margin, may be constructed and placed across the foot of the bed. The patient is instructed to put each heel into the cups seriatim, or a flat board perforated with a number of holes may be placed upon the bed, and the patient encouraged to place one foot and then the other into each of the holes. The movements are gradually increased from such simple procedures as have been described to more complicated forms; such are tracing with the toes the outlines of squares, circles, or zigzags drawn upon the floor, walking over small hurdles forwards, sideways, and backwards, and a series of exercises which may require the assistance of parallel bars, by which the patient supports himself with the aid of his hands.

These are some of the devices elaborated with great care by Fraenkel, but the physician or the patient himself may readily devise others suitable to individual cases.

THE lectures at the Hospital for Consumption and Diseases of the Chest, Brompton, commence on Wednesday, May 22nd, at 4 p.m., with a lecture by Dr. Mitchell Bruce on the "Diagnosis, Prognosis, and Treatment of Quiescent Tuberculosis."

A CASE OF MULTIPLE SUBCUTANEOUS SARCOMATOUS TUMOUR.

Demonstrated* by

JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

Here is a healthy-looking man of about forty years of age who shows a number of large tumours about his chest and abdomen. The history he gives us is that ten years ago he had a lump in the right groin. He says that for the past six months that lump has been getting bigger. So you see it has been quiescent for ten years, and then has burst into activity and enlarged. Here is another higher in the abdomen, another higher still, one on the shoulder, and another on the other side of the abdomen. These are all very definite indeed, and some of them are quite large. You see I can pucker the skin up over the growths. One has become adherent to the skin, and there is a plum-coloured congestion over it. I find another lump in the middle of his pubes which is not a gland. Clearly the condition has spread by the lymphatics. He says he feels well, and has no pain at all; in fact, he does not know when new ones come except by feeling them. He is not a married man, and tells us that ten or twelve years ago he had some venereal disorder. I do not know whether it was due to that venereal disorder, but he says he got the lump in the groin subsequent to that disorder. One finds that enlargement of the glands after syphilis does not entirely subside after the attack, and perhaps this man's did not recede, but took on an abnormal process. That is a common history in lymphadenoma, that after enlargement of a gland in sore throat it remained so for years, and then took on greater enlargement. This occurs in any structure which has been chronically inflamed. His mother died at seventy-five with cancer of the liver. The father is said to have died from internal hæmorrhage, possibly the result of strain. We do find transmutation in inherited transmission, transmutation from one type of growth to another. I should call this a case of multiple subcutaneous sarcomatous tumour taking origin from the glands. There is no tendency to enlarged glands in the neck. That man will get thinner from competitive nutrition; the new tumours and tissues will take up the nutrition required for the body.

* At the Medical Graduates College and Polyclinic.

Digitized by Google

I am asked as to treatment. I am afraid I should say it does not matter in the least what one does; no treatment succeeds in curing the disease. It is not a case for any operation whatever. The infective material is already widely diffused in the lymphatic system. If we were to cut one tumour out, another would come, and there is no stopping it. Moreover, the infective material may be already in his blood, but I think it is likely that the spreading cells are not in his blood yet, but that they are in the lymphatic spaces. I expect that in a few months that man will be covered by tumours, and that in a very little time his health will begin to fail. I would give him arsenic; there is nothing but that to arrest the progress of the disease, or to slow its onset. It should be given in very liberal doses, six or eight drops of Fowler's solution three times a day, until it definitely disagrees with him. In the Museum there is a portrait of a young surgeon who had these subcutaneous tumours, in whom the disease ran a singularly rapid course, so that he died within six months of the first appearance of the tumours. I show you some pictures from the 'New Sydenham Society's Atlas.' The disease generally begins in the tonsils, and the infective matter travels out to the lymphatic glands, and produces similar growths elsewhere. It tends to run an infective course, malignant in so far as it is infective, but not tending to spread to different kinds of tissue. Sometimes it may affect the skin, but usually it keeps definitely to the lymphatic system. Having begun in the tonsils, it travels down into the axilla, and also from the neck down into the thorax, thence into the mediastinum, down into the abdomen, and into the lymphatic glands about the aorta and the large vessels of the spine, where there may be a congeries of growths. Finally it reaches the groin, causing a bubo. Then it produces disease of the spleen, and brings about death by emaciation. These cases often die suddenly from heart failure. Here I show views of tumours from a patient whose case is in Sir Robert Carswell's 'Atlas,' and it was shown by him to Hodgkin, who said at once, "That is my disease." There are large masses shown growing in the substance of the spleen. If such lesions are found in the liver, people generally say it is a mass of soft cancer; indeed, it had been recorded by a Paris physician, under whose care the patient was, as bleeding cancer, but we have Hodgkin's recognition of it as an example of his disease, and there is nothing definitely cancerous about it. In this case I can get no history of cancerous inheritance.

We saw behind the screen a girl with a somewhat similar condition, and here, again, there is no history of cancerous inheritance; but her mother was said to have died of bronchitis after a long wasting illness and very profuse expectoration. So we must ask whether it was likely to have been phthisis or chronic bronchitis. You saw her sister here, a rather delicate-looking woman, who said she was in good health, and had no cough. The girl is the youngest of fourteen children, eleven of whom are living, and none of those eleven have had any special symptoms. The father is still living. If the mother had developed some tubercular disease late in life, the youngest child is the most likely to have inherited it. Instances of cure by arsenic in these cases have been published, but in my experience all the cases have relapsed eventually. I have seen the severity of the condition very considerably diminished by the administration of arsenic. A gentleman present mentions cacodylate of soda, which I have not tried, but which I think might be tried in this case.

The Physiological Care of Colds.—C. H. Shepard considers that colds come from within. For treatment he gives the following advice: To make a radical cure of a cold let the patient abstain entirely from food for at least twenty-four hours. Should the bowels be at all inactive, it is desirable that they be thoroughly flushed with warm water. He should drink freely of pure water, taking a brisk walk in the open air, and then a Turkish bath. This may well be followed by an oil rub, sometimes called a Roman bath, which mollifies the skin, making it more flexible and active. The patient will find a permanent benefit therefrom. The internal pressure will be relieved, and the cure perfected. Necessarily chronic cases need more persevering treatment, but each day will show progress, and only perseverance is needed to triumph ultimately over the morbid action.—*Medical Record*, April 27th.

Digitized by Google

THE PRACTICE OF BLOOD-LETTING.

By J. F. BRISCOE, M.R.C.S.

I HAVE previously* given an account of the past and present of blood-letting, methods of blood-letting, and conditions indicating blood-letting; I now proceed to attempt a classification, illustrative of conditions of the system in which venesection is required. The pathological conditions of the system indicating venesection are those of a non-inflammatory nature, or those affections which arise from the secondary or tertiary consequences of inflammation. The principal object of blood-letting for general purposes is to relieve asphyxial symptoms, and the failure of the heart's action which arises therefrom. It is indicated in perilous states of the various systems of the body. In its action it relieves congestion, counteracts backward pressure of the blood, and in toxæmia removes effete products from the circulatory system. It is a therapeutic agent with an immense field of action when it is timely and wisely practised. The indications for its use can only be established at the bedside, and the so-called physiological effects of withdrawal of blood can only give an imperfect idea of its therapeutic effects. It reinstates cellular oxidation and thereby encourages the action of medicinal agents. As a specific remedy in the strangulation of the blood and tissues, its action, as it were, closely corresponds to the operation of herniotomy; for it not only relieves congestion and pressure, but is eminently a restorative in impending death. A convulsive seizure may at any time seriously arrest the pulmonary and circulatory systems and terminate life. At the post-mortem, which is generally made twenty-four hours after death, we are frequently unable to verify the distended right heart of asphyxia. The evidence of this acute distention is frequently a mere conjecture, and not often substantiated by the unaided eye.

A point worthy of great consideration, when seeking for congestion or hæmorrhage into an organ after death, is to examine first that particular organ. For instance, it is obvious that if punctiform hæmorrhages or congestion are present in the brain, they would possibly escape notice if the thorax were opened previously in the usual routine of the post-mortem room; the large vessels having been opened in the chest, the cerebral cavity would be drained of its blood. This is verified on suffocating poultry, for the congested cerebral vessels can be drained when the post-mortem examination is made, by first severing the large vessels of the chest.

I shall endeavour to arrange as well as possible the subject matter of this paper, and shall attempt to classify conditions illustrative of the practice of blood-letting. Of the convulsive seizures deserving our attention we will consider tetanus, hydrophobia, and puerperal, uræmic, and not least, the epileptic forms of eclampsia. At any time the tetanoid state may interfere with the respiratory act, and thus blood-letting may be necessary to avert suffocation. Again in hydrophobia, venesection to syncope has been tried. In puerperal convulsions, venesection is indicated when there is plethora and much arterial tension. It has been a form of practice which has proved of great benefit in the hands of some practitioners. I once had a patient whom I bled before she was delivered, and the case did well. However, if it is likely that the woman can be rapidly delivered with forceps, venesection should not be practised, unless convulsions reappear after the placenta has been removed. Dr. J. G. Swayne, in the 'British Medical Journal,' April 4th, 1863, records eleven successful cases of genuine puerperal eclampsia. Of these, nine were *primiparæ*, and two *multiparæ*. He says, "This corresponds with what has been generally observed: viz. that the great majority of cases of puerperal convulsions are *primiparæ*." In uræmia there is no doubt that venesection clears the circulation quicker than diuretics. Blood-letting, therefore, from seven ounces upwards, is sound practice for the nervous symptoms which arise in the course of acute or chronic Bright's disease. Again in the *status epilepticus* venesection is required, but not as a routine practice. I cannot believe, as I have heard stated, that you may bleed an epileptic to death and not a drop of blood will leave the head. If this is so, it would seem that convulsive seizures are very much ruled by the sympathetic nervous system. We are told by Dr. Savage "that, in the insane, I cannot say that I have seen insanity affected, except for the time, by the administration

* Vide 'Clinical Journal,' August 9th and September 20th, 1899.

Digitized by Google

of anæsthetics; the action of chloroform in the *status epilepticus* is only *pro tempore*." Moreover, in the last stage of asphyxia in epilepsy, nitrite of amyl would be useless; the systemic arterial system having been exhausted and become anæmic, and the right side of the heart distended to its fullest extent, there follows the importance of venesection in the *status epilepticus*.

This lends itself to our imagination, and might help us in theory to expound the asphyxial problem in convulsive seizures. If epilepsy, as Sir Samuel Wilks thinks, is central, *i. e.* due to molecular upset of the nervous elements, possibly of a toxæmic nature, the very beginning of an epileptic fit would appear to simulate an asthmatic seizure. The fact that spasm of the arterial system is relieved by nitrite of amyl further throws light on the matter. If it is a fact that the irritation of teething, worms, or other causes may upset the stability of an impressionable nervous system, then we have no difficulty in solving the problem. However, a toxin circulating in the nervous tissues is nowadays recognised as causative of mental disorders, interfering with cellular metabolism, and resulting in the death or the deterioration of areas of nervous tissue. Toxæmia may be evanescent, acute, chronic, or persistent; and hence the curability of certain forms of mental disease, after the physical affection has been removed by suitable treatment. The pathology of many diseases can be explained in this way; for instance, is not anæmia possibly encouraged by the circulation in the blood of an intestinal gas, such as H_2S, for it is known that sulphurous acid if inhaled produces decomposition of the hæmoglobin? The large amount of iron which we administer in chlorosis may not so much affect the corpuscles themselves, but by combining with the H_2S, in the intestinal canal of the chlorotic girl, may render this gas inert in the system, and escape with it in the fæces as sulphide of iron. Again, CO combines readily with hæmoglobin, and is a very deadly poison in colliery districts. And we know of other substances equally deleterious to the vitality of the corpuscle. The physiological chemist has much to unravel with regard to the nature and action of toxic agents, and without his assistance the real foundation of disease will never be revealed by the pathologist if toxins are of such moment. But who can say to the contrary, that in the near future many diseases will be traced to the intestinal canal, and that fæcal analysis will become an essential feature of clinical study? It is obvious that whatever is the nature of the toxin in mental disorders, it must be variable in dosage, and must affect the nervous tissues differently in the many forms of mental disease. It is conceivable that in epilepsy there is a bad habit of metabolism, but the nature of the toxic agent, if there be one, is far from having been ascertained. We need a more thorough acquaintance than we already possess of the chemistry of the intestinal tract, Then there is the chemistry of the toxins which we suppose may arise from the products of the microbe; and lastly, the toxins produced by disturbed metabolism from exposure to cold or other exciting agencies. Each toxin would appear to have its own chemical exit from the system, and there are those poisons which are influenced by the open-air treatment; but failing anti-toxins and oxygenation, it appears to me that we have beyond the skin, the kidneys, and the exit from the bowels, another important means of elimination, namely, venesection. Later on we shall see that blood-letting encourages corpuscular activity.

If it is a truth that there are ganglia which regulate the co-ordination of movements, then we have something tangible when we consider nervous affections. At any time the irritating body within the economy may so disturb the nervous apparatus as to produce unsoundness of mind. The ordinary sphincters of the body are ruled by the nervous system, and the arterial system is exposed to the same influence. For instance, the vaso-motor system rapidly influences the arteries in the case of facial or other flushings on the exterior or interior of our bodies. Sir Crichton-Browne has recorded several cases in which nitrite of amyl has cured the *status epilepticus*. Yet we should bear in mind the action of nitrites on the blood. There is so much conjecture and uncertainty with regard to epilepsy and its causes, that the recommendation of nitrite of amyl is worthy of all consideration. But, failing relief by this drug or anæsthetics in the *status epilepticus*, the congested venous system should be at once relieved by venesection, otherwise the patient may die from asphyxia.

An illustrative case bearing on this matter is reported in the 'Lancet,' September 22nd, 1900,

Digitized by Google

by Dr. W. T. D. Allen, Resident Medical Officer, Liverpool Workhouse Hospital:

Trephining and drainage in an apparently moribund case of status epilepticus; recovery.—The patient was a woman aged thirty-six, admitted into the Liverpool Workhouse Hospital. The epileptiform convulsions appeared for the first time at thirty-six years of age. She was addicted to intemperance, and was admitted suffering from severe epileptiform convulsions. She recovered from this attack and resumed her occupation, but four months after the convulsions returned again. This time the patient was semi-unconscious, and during twenty hours had over forty fits. Each fit lasted two or three minutes. She appeared to be moribund, and Dr. Alexander, under whose care she was, trephined over the upper motor area on the left side. The dura mater bulged into the opening; the cerebral veins were much enlarged and lay bathed in a layer of fluid a quarter of an inch thick. The fluid was held in position in the meshes of arachnoid and pia mater, and when the meshes were pricked the fluid ran away in a clear sparkling stream. There were no signs of any gross lesion such as cerebral tumour, or bone injury, or disease. After the operation the patient had several fits, but she gradually recovered from them; the wound healed, and the patient became quite well and strong.

Remarks by Dr. Alexander:—"In the presence of deep unconsciousness in *status epilepticus* the medical man often feels quite helpless. Bleeding is often useless, because the blood will not flow, and oxygen or chloroform makes no impression. In such cases the compressed cerebral hemispheres may be relieved, either, as in this case, by drainage of the serous effusion that sometimes exists, or, in others, by the venesection of the congested cerebral veins. The operation itself, under aseptic precautions, is so harmless that its performance is more than justified under such serious conditions as the above case presented."

Dr. Mott has brought before the notice of psychologists certain changes in the brain, muscles and other organs, found in persons having died with prolonged epileptiform convulsions. Those of us who have had the opportunity of seeing his microscopical sections thrown on the sheet from the lantern, will be at once struck with the bearing they have on the present subject. He showed us marked changes in the chromatic substance of the cortical pyramidal cell and also œdema of the perivascular and perineural lymph-space. Besides this, he states that there is venous congestion with arterial anæmia. Are not these microscopical appearances, brought about by repeated attacks of congestion, the outcome of mechanical pressure: and may not asphyxial toxic changes add their share of influence in producing these appearances? Thus such a condition points to venesection in preference to nitrite of amyl. It would be interesting to know whether similar pathological appearances in the brain-cells which Dr. Mott has described, and which he would teach us are peculiar to the brain of the person dying after prolonged *status epilepticus*, are not also to be found in brains of persons other than the epileptic and as a consequence of asphyxial changes. I ask this question very pertinently from one to whom the Psychological Association is very much indebted. For instance, in persons dying after severe attacks of mania, acute delirious mania, tetanus, hydrophobia, and from ordinary lighting-gas suffocation, etc., it is obvious that mechanical pressure and toxic changes play a very important part pathologically. Pathological microscopy would appear as contradictory sometimes as the multitudinous forms which micro-organisms exhibit. The following case came under my notice many years ago and is worthy of careful consideration, inasmuch as Dr. Mott states that in three cases of death in young persons during the *status epilepticus*, death ensued from asphyxia and dilatation of the right side of the heart.

W. W—, æt. 27, was removed from the water from a depth of at least thirty feet. He was wearing a diver's dress and was searching alongside Sandridge Pier, Melbourne, for a large bottle of quicksilver. No air coming to the surface of the water, the attendant dragged him up. He was apparently dead, when I saw him about three minutes after, lying on the pier. I at once laid him across an extemporised pillow in the supine position, and practised the Howard method of artificial respiration for two minutes. As he did not respond to artificial respiration, I quickly diagnosed that he was suffering from the final stage of asphyxia. I could not get a pulse anywhere although distant hurried faint heart-sounds, as in pronounced suffocation, were heard

Digitized by Google

when I applied my unaided ear over his bare chest. There had been no voluntary respiratory act from the time I first saw him; his body all over was of a dusky leaden hue; his pupils were widely dilated and conjunctivæ insensitive, and he was lying on the pier as if the candle of life were out. When I decided to bleed him, I pictured in my mind a distended and paralysed right heart with a backward current, and the treatment I adopted fully confirmed the teaching of Sir Samuel Wilks on Blood-letting. Had I continued artificial respiration any longer, another minute or so would have seen his end, for, as was shown subsequently, his tissues were grossly suffused with extravasated blood. I asked an onlooker for a knife and opened a vein in the right wrist. At first little blood escaped, but continuing the shampooing of the extremities, which was being adopted, soon dark venous blood trickled out of the incision wound, and later on it spurted. When about fifteen ounces of blood had escaped on the pier the patient began to move his limbs; and to our great surprise he gave an involuntary respiration. This I at once imitated by artificial means. Thirty ounces or thereabouts of dark blood had already left his venous circulation, so I bound the wound up with a handkerchief. Needless to say, during this momentous period, all the adjuvants necessary for the resuscitation of those suffering from suspended animation were employed. The patient having so far recovered was removed to his house two miles away. He was placed on a soft bed without a pillow and the chamber darkened. For the first three days he lay in bed moving his limbs but slowly. On the second day he evacuated a bloody motion, and he also passed blood with the urine. The surface of the body was covered with a petechial eruption. On the third day he gesticulated in a drunken fashion. He was quite blind, so the eyes were carefully examined with the ophthalmoscope, and in both there were extensive and defined retinal hæmorrhages. As he was unable to appreciate the bright light from the mirror, the exploration of the interior of both globes was readily accomplished. The patient was apparently deaf. On the fifth day he shed tears, and seemed to recognise his mother; he also drank freely of milk, which, up to the present, had been his diet. On the sixth day he spoke to me and was conscious: this was the first return of intelligence from the time I saw him on the pier. However, he remembered nothing of the accident, either before or after the event. He was still able to distinguish light from darkness. Blue glasses were procured for his use. Up to this date he had been taking two grains of calomel twice daily, which I now stopped. He made an uninterrupted recovery, complaining at times of pains in his limbs and body. The urine gradually cleared itself and the motions contained no blood. The petechial eruption rapidly disappeared. In three weeks from the misfortune the eyesight became normal, and he was out on the door-step at the end of the month. The pathological importance of this case is very interesting and remarkable, and are we not justified in assuming that every organ and tissue of his being was more or less affected with extravasated blood? Was this the result of an epileptic seizure? The young man was at the time fulfilling his father's duty, who was ill. He had only descended into the depths of the water on two previous occasions. There were known to be sharks in Hobson's Bay. Further he was of an anxious temperament. Although there was some doubt as to the working of the machinery, there was no real evidence that the diving dress had gone wrong. It was assumed that one of the valves was faulty, since no air was coming to the surface of the water before he was pulled up. But, if he had a fit he would have fallen and so perhaps been buried in the mud. The father, who had been the diver for Hobson's Bay, Melbourne, for several years, told me that he had on several occasions experienced vertiginous attacks when at the bottom of the water. This, too, I believe is the experience of other divers, and results from great pressure of water. But supposing it was only a case of simple asphyxia, is it not equally astounding that the case terminated in recovery, and will it not materially support my views of blood-letting when I describe later on its judicious practice in gas-poisoning. Had I persisted in artificial respiration for another three or four minutes the patient would have died. The man's venous system had burst, and was leaking in many places as it was full to repletion, while his arterial system we assume was more or less empty. I relieved him by venesection, and set the pendulum of life swinging again. This case illustrates very forcibly the remarks of Sir Samuel Wilks in one of his cases, when he says "her life was as clearly saved by the bleeding as if I had dragged her out of the water; and this is more than I can say from the use of drugs."

(To be continued.)

Digitized by Google

CREOSOTAL (VON HEYDEN) IN SEVERE SCROFULOUS AFFECTIONS AND OZÆNA.

DR. MAZZAROTTO, Director of the City Hospital at Rome-Raciglione, has obtained excellent curative effects with creosotal in all forms of tuberculosis. In pulmonary tuberculosis a single month of the treatment produced a marked improvement in the appetite and the general condition, caused the fever and night-sweats to disappear, and diminished the cough. After four months of treatment the percussion and auscultation sounds were greatly improved, and there was an increase of weight of from $6\frac{3}{5}$ lbs. to $13\frac{1}{5}$ lbs. In one case, treated for eight months, there was an increase of from $105\frac{3}{5}$ lbs. to $136\frac{3}{5}$ lbs. There were patients among these who had not only taken courses of creosote pills without advantage, but had suffered serious damage of the digestive organs as a result. These experiences led Mazzaretto to employ creosotal in scrofulosis and affections of the lymphatic glands. Based upon the records of twenty cases, he describes unexpectedly good results, such as he had never seen from other treatment. None of the patients had nausea or irritative symptoms of any kind; and there were some amongst them whose digestion was in extremely bad condition. Every fifteenth or twentieth day the treatment was interrupted for a few days. The urine of these patients never contained albumin or any other abnormal constituent during the treatment; the colour of the secretion sometimes remained normal and sometimes became brownish; and it always smelled of creosote after a few days' administration of the creosotal. The patient's breath also smelled of creosote, a sure sign that the creosote derived from the creosotal was circulating in the blood, and being excreted by the respiratory organs. Mazzarotto administered the creosotal in syrup and cod-liver oil, beginning with three drops three times daily, and increasing the daily dose by three drops every three days until twenty-four or thirty drops were taken three times daily.

Mazzarotto believes that the success of this method of treatment in ozæna is a new proof of the scrofulo-lymphatic basis of this disease. While nothing was attained by local, or even surgical means, surprisingly rapid cures followed the use of simple cleansing and hygienic methods, provided their effect was supported by the use of the anti-tuberculous and anti-scrofulous remedy, creosotal. Almost all the cases of ozæna that he has treated had complete anosmia with marked hypochondriasis and mental depression. The active agents that cause ozæna are very common in scrofulous individuals. He believes all the multitudinous proposed remedies for ozæna to be useless, or even harmful; they are always troublesome and painful for the patient. Cleansing injections, together with the internal administration of creosotal, suffice to bring about a marked and usually rapid betterment in the local infectious process. Ozæna is a localised manifestation of the scrofulous tendency, which in general is favourably influenced by creosotal; hence its remarkable action in ozæna. Mazzarotto's opinions, in his own words, are, "The remedies are at hand, and medical science is neither poor nor is therapeusis in its infancy; but perseverance, patience, and study are required to combat the scepticism, the mistrust, and the apathy that is so prone to possess the mind after a professional disappointment."

In the case of a girl, twenty-one years old, who had had a purulent otorrhœa and ozæna for eight years, the treatment followed was abundant injections with the Weber douche, at first with a lukewarm boric acid solution containing three grains of naphthol to the quart, and later with a solution of bromine and iodine salts. The administration of creosotal was begun on the third day, five drops being given three times daily, and gradually increased to twenty-five drops thrice daily, and administered in syrup. The fetor disappeared fairly quickly, and after six months of the combined internal and external treatment, Mazzarotto obtained so favourable and brilliant a result that he now proclaims himself an enthusiast for the internal treatment of this disease with creosotal. In another scrofulous patient, with an ozæna of three years' standing, a post-auricular impetiginous eczema and enlarged cervical glands, a complete cure was effected by seven months of the same treatment. A rachitic boy, fourteen years old, with an extensive tubercular ulceration of the skin, covering the entire right cheek, the left side of the neck, and the back of the right hand, was treated externally, after the destruction and extirpation of the entire affected surface, with iodoform, whilst the internal treatment consisted of the administration of creosotal in doses of from five to forty drops daily in syrup. There was a complete cure in three months.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 448. WEDNESDAY, MAY 29, 1901. Vol. XVIII. No. 6.

CONTENTS.

PAGE

*With Dr. Hale White in the Wards of Guy's Hospital 81

*A Note on the Diagnostic Value of X-ray Photography in Osteo-arthritis. By E. W. H. SHENTON, M.R.C.S., L.R.C.P 85

*Four Clinical Lectures on Some Affections of the Kidney. By A. H. TUBBY, M.S., F.R.C.S. Lecture I 86

*The Practice of Blood-letting. By J. F. BRISCOE, M.R.C.S. (*continued*) ... 90

Mr. James Berry on Dyspnœa caused by Goitre 96

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, etc., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications, with the exception of those relating to Advertisements, should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 1s. 6d. each, or will be forwarded post free on receipt of 1s. 9d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

WITH DR. HALE WHITE IN THE WARDS OF GUY'S HOSPITAL.

HERE is a man æt. 44, who comes with fluid in his abdominal cavity. Do you think there is ever any difficulty in ascertaining whether fluid which you feel is really in the peritoneal cavity or not? There is rarely much difficulty in the case of men, but in women there is often extreme difficulty in that respect. By no means infrequently there comes into the hospital a female case in which various people who examine the patient differ very much in their opinions, that is to say, whether it is ascites, or whether it is an ovarian tumour. If an ovarian tumour gets so big that it drives the intestines upwards and backwards it is very difficult to tell what the condition is, because by the time you see the patient the abdomen is so distended by the tumour that you cannot say whether it is fluid in the peritoneal cavity or not. In such cases often the swelling is no longer more one side than the other; it seems to fill the whole abdominal cavity, and the patient has sometimes forgotten which way it grew up. You might think that what is told you in the books might help you, namely, comparing the measurements from the umbilicus to the ensiform cartilage, and from the umbilicus to the pubes. But last time I took charge of Miriam Ward there was a woman who was seen by a surgeon, and by myself, and by the clinicals, and house physician, and the opinion was perfectly unanimous; there was no dispute, and the opinion was that the patient had an ovarian tumour. Measurements were taken, and the clinicals made a very accurate report, and everyone was satisfied. Moreover, the tumour seemed to lie a little more over to the left than to the right, and the surgeon accordingly decided to operate. On opening the abdomen no tumour was found, but what was found was a condition of ascites. That made us especially interested in these measurements which are referred to in the books, and therefore many comparatively healthy women in the ward were measured, and some

Digitized by Google

ascites cases were also measured, namely, from the umbilicus to the ensiform cartilage. If the measurements meant anything, they meant that one third of the women in that ward had got ovarian tumour. The conclusion which was come to was that this measurement as an aid to diagnosis is valueless, and I do not think anyone need really attach any importance to it. I do not know any means of ascertaining whether a swelling of that kind in a woman is an ovarian tumour, or whether it is ascites. Vaginal examination does not help you very much, because very big ovarian tumours rise up out of the pelvis. Often the bigger the ovarian tumour is, the less of it there is to be felt in the vagina, because it is too big to come down into the pelvis. So in women you have always to be on your guard. In another case, which was described not long ago in the 'Guy's Gazette,' the house physician thought the patient was suffering from ascites when he saw her, and accordingly took her into the general wards of the hospital. Someone else who saw her thought the swelling was an ovarian tumour, and transferred the patient to the obstetric ward, where it was operated upon and found to be ascites. The books tell you to notice whether the distension grows upwards from the pelvis, to see whether it is on one side more than on another, and, indeed, on reading the books one would think that the man who made a mistake was very stupid; but there are cases in which the best observers are wrong. In these days of antiseptic surgery less harm is done than formerly if you make a mistake; indeed, many people think that the best treatment you can adopt for extreme ascites is to let the fluid out by an abdominal incision. The case in Miriam I first spoke of was perhaps more instructive than I have made clear, because she had a murmur, and so we recognised all along the possibility that she had ascites, due to the condition of her heart; and yet the unanimous opinion was in favour of an ovarian tumour. The ascitic fluid was all let out by the operation for the supposed ovarian tumour, and she did very satisfactorily, and was much benefited by the removal of the fluid. But of course a direct diagnosis may matter very much from the point of view of your reputation.

Well, this man has got a quantity of fluid in the abdomen. By rolling him over you have found that the part of the abdomen previously resonant becomes dull, and *vice versa*. Do you find always in the case of ascites that this is so? It is not always so by any means if the patient has peritoneal adhesions. By far the most difficult cases of all to diagnose are those where there is so much peritonitis that the fluid is confined in spaces formed by the adhesions, and then it may be that no amount of shifting alters the resonance. In this case, when you turn the man over the upper flank becomes resonant; when you shift him back it is dull; you also get a thrill; and, taking all the facts together, there is no doubt fluid in his abdomen. In such a case as this what do you think is the first thing to do to the man in the direction of finding out what it is. You will, of course, proceed to see what disease it is by the process of exclusion. You will examine his heart and chest and lungs, and you will also examine his urine and his pulse and his eyes, to see whether the condition may be due to Bright's disease. This will enable you to quickly put some of the possible causes out of court. We have been over those possibilities here, and we have decided that the condition is not due to anything in the chest, and that it is not due to Bright's disease. We have narrowed the diagnosis down to there being some local cause in the peritoneal cavity for the ascites. Now, what are the local conditions in that cavity which will give rise to ascites? There are the various forms of chronic peritonitis. I will not say anything else, though I do not think that would do for the books: most books would expect you to say cirrhosis of the liver. The evidence which we have goes to show that when a man with cirrhosis of the liver gets acites, it is, strictly speaking, not due to the cirrhosis of his liver. From reading you get the impression that it is because of the fibrous tissue in the liver contracting upon the portal vein, and that consequently the man gets fluid effused from the periphery of the portal vein, and so he gets ascites. But I think there is not the least doubt that this is not the whole explanation, and for these reasons. In the first place, you cannot produce ascites in animals by aseptic ligature of the portal vein. Surgeons often ligature veins in man, but we do not find œdema as the result of it. If the blocking a large vein by the ligature could produce œdema, what a serious thing ligature of veins might be. If the femoral vein was wounded and bleeding fast, the surgeon would not hesitate to tie it, but

Digitized by Google

that would not produce œdema of the leg. Then again, very often in cirrhosis there cannot be very much increased portal vein pressure, because of the anastomosis of the other veins, chiefly the hæmorrhoidal and the œsophageal. Another point is that if you look carefully at the peritoneum you will very often find in cirrhosis of the liver that the peritoneum is opaque and obviously diseased. So it is highly probable that in cirrhosis of the liver something goes wrong with the peritoneum of the abdomen, in consequence of which it secretes a great deal of fluid, just as something goes wrong in the peritoneum of the abdomen in cases of Bright's disease, in consequence of which it secretes a quantity of fluid. Another point is that you may get ascites long before the liver has begun to contract up, and therefore before the fibrous tissue would press on the portal vein. When I have made post mortems I have often looked to see if I could make out that the portal vein was dilated in cirrhosis of the liver, but I never saw the slightest evidence of it. One of the results of cirrhosis is probably to lead to such a condition of the endothelium of portal capillaries as to permit transudation.

Of late years it has been said that the right way to treat ascites which is associated with cirrhosis of the liver is to try to produce adhesions between the liver and the diaphragm, so as to help the circulation between the portal system and the general system. Well, in the first place, it is an operation with a high mortality. In the next place, of the people who have had that done some have not been permanently benefited. You must remember that the diagnosis of cirrhosis of the liver, as we know to our cost where we have a large post-mortem experience, may be wrong, so that possibly in the few cases where the treatment has appeared to succeed it may have been that the disease was not cirrhosis. Then, again, it may be impossible to produce adhesions. Mr. Fripp did this operation the other day on a patient in clinical ward who was not at all benefited by it, for he died not long after. The post-mortem showed undoubted cirrhosis, but no adhesions had formed between the diaphragm and liver. It is noteworthy that, although there was much ascites, Mr. Fripp was at the operation struck by the fact that the gastric veins that anastomose with the œsophageal were not much dilated, and this is very important, for it supports my contention that the outpouring of ascitic fluid is not solely due to increased portal pressure, and it also points to the fact that but little benefit was to be expected even if adhesions had produced a free anastomosis between the portal and general venous systems.

In this man now in bed before us we have no evidence that his ascites is associated with cirrhosis. We cannot feel the liver to be cirrhotic; he does not look like a sufferer from cirrhosis; he has shown no symptom of it except ascites, and he is a teetotaller. So we have in his case to think of chronic peritonitis apart from cirrhosis. What are the common causes of chronic peritonitis? I think tubercle is the commonest, then the disease called simple chronic peritonitis, and then malignant growth. Here we have no evidence that the trouble is tubercular. Of course it may be what is called simple chronic peritonitis, but against it being that is this fact: in the cases of so-called simple chronic peritonitis you are very likely to get the peritoneum matted very much, so that you can feel lumps of matted peritoneum about; you can feel the omentum forming a transverse tumour in the belly. Bright's disease is often associated with such a condition. We cannot find any evidence of that here. On the other hand, the man looks very wasted, and strikes one as possibly having malignant disease. So at present I think the diagnosis of malignant peritonitis holds the field. He has such a sunken wasted look that he has probably wasted very rapidly, and therefore it is quite possible that his malignant peritonitis is secondary to malignant disease of the stomach, for if that disease causes much indigestion the wasting may be very rapid. [This view was proved to be correct, for the abdominal distension increased to such a degree that paracentesis was necessary, and when the fluid was drawn off it was easy to feel a large mass occupying the greater curvature of the stomach.] The treatment must be palliative, because if this man's ascites is due to malignant disease it is clear that he is beyond the range of operation. The only form of abdominal malignant disease upon which you can operate with a view of cure are those which you diagnose early. Occasionally triumphant cases occur in which malignant disease in the abdomen is diagnosed sufficiently early for the growth to be excised, and in such cases it is usually in the pylorus, for growth there

Digitized by Google

soon gives marked symptoms, because of the gastric dilatation that often ensues; but such cases are very rare. Although unfortunately abdominal malignant disease is common, yet personally I have never had a successful case of excision, although several times at my request a surgeon has opened the abdomen. In every case where he has done so either the growth has been impossible to remove, or there have been extensive secondary deposits. But he can often do much good by short-circuiting past the growth.

The next patient is a man æt. 49, and his case is one of some interest. We came to the conclusion that he had not had rheumatic fever. He had an attack of rheumatism seven years ago, but there is no evidence that it was genuine rheumatic fever. Now he comes in here with pains about him, especially in the joints. When he first came in the question was raised whether it was rheumatic fever this time. The arguments against the present trouble being rheumatic fever are as follows: first, the pain has not flitted from joint to joint; it has kept in the same joints. His temperature has never been up since he has been in; there is no heart lesion, and there has been no marked sweating. Look at his legs. You can see that his right leg is considerably more wasted than his left, and by actual measurement we are told the difference is one and a half inches in circumference. The importance of that in the argument is that it shows the joint disease has lasted some little while, as the man tells us. What he has got is arthritic muscular atrophy due to disease in the right knee and right ankle. Arthritic muscular atrophy is a condition of atrophy of the muscles, which comes on in people who have joint disease, quite independently of the cause of that joint disease; it can be produced in animals by causing artificial joint disease, but it does not occur if previously the posterior roots have been cut. So it is quite clear that the muscles are in some functional way associated with the joint they move, and that this functional association is controlled by the cord. That association is so close and intimate that when the joints are affected the muscles atrophy. Of that this is a very striking instance, and as the joint trouble must have lasted some little time to produce marked arthritic atrophy, and the trouble in rheumatic fever flits from joint to joint, we are able to make out that he has not had rheumatic fever, and his disease is most likely to be chronic rheumatoid arthritis. Two other things pass through our mind, and one is whether it is gonorrhœal rheumatism. There is no evidence of that. The other possibility is that it might be gouty, and in favour of that view he says that when he first came to the out-patient department he was told that he had gout. Very often it is extremely difficult to tell whether joint disease is rheumatoid arthritis or gout. His pain has been relieved by salicylates, but you must not assume from that that it is genuine rheumatic fever; very often the pain of rheumatoid arthritis may be relieved in the same way. I have brought some skiagrams for you to see; they are taken from the woman in 14 Bed in Mary Ward, whose case was described in the 'Clinical Journal,' for December 12th, 1900, and who has that rare condition of acute rheumatoid arthritis of young adults,—for remember rheumatoid arthritis is usually a chronic disease. The acute disease is accompanied by a temperature often considerable. It affects younger people, it affects many joints, but typically the phalangeal joints, and the condition is very intractable. There was in this girl extreme muscular atrophy. You can see that the skiagram taken by Mr. Shenton shows very beautifully what, as far as I am aware, has not been observed before; you notice that the ends of the bones in the neighbourhood of the joints appear particularly transparent. I also show you a skiagram of a normal hand, for comparison with the other. The difference between the two pictures is very striking. They appear to teach that in acute rheumatoid arthritis, long before there is any outgrowth of bone (which rarely occurs in these cases) the heads of the bones are abnormally transparent to the X rays.

If a private patient came to you with chronic rheumatoid arthritis, like this man, what would be the first thing you would do for him? I think the best treatment for rheumatoid arthritis is undoubtedly local applications of heat; and that is why Bath and Aix-les-Bains are so popular, because there the patients apply plenty of hot water externally. People seem often to do better at Aix than they do at Bath, but why that is I do not know. It may be that there is some difference in the climate. At both places much of the good is due to the excellence of the bath assistants; they shampoo, douche, and mas-

Digitized by Google

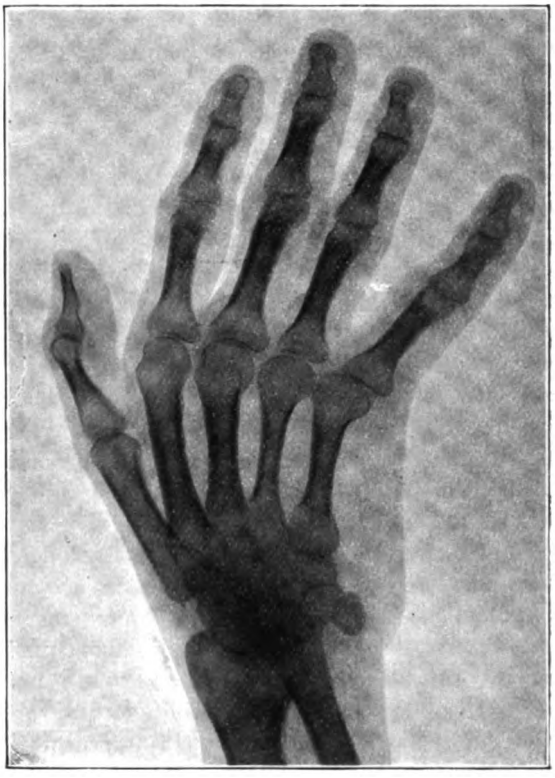
sage the patients very thoroughly and systematically. This patient tells us he got well of other attacks at Bath, which tends to confirm our diagnosis. If you want to send a patient away for benefit in summer you may send him to Aix, but you may send him to Bath all the year round. The objection to Bath is that some find it enervating. An excellent place for these patients to go to in the winter is Helouan, in Egypt, though, of course, it is a long way. The warm, desert air of Helouan does much good, and there are sumptuous hot baths. Nowadays a good way of applying warmth locally is by means of the electric light. And it is by far the cleanest way. A sort of cradle is put over the whole body or over a single limb, and over the cradle is put an asbestos sheet to keep in the heat. Inside the cradle are several electric incandescent lamps, by means of which you can get up a temperature of 300 to 400 F. But the worst of rheumatoid arthritis is that you can never be quite sure that what you are going to try will do good. You will do well to always begin treatment by the local application of heat and massage. But very often you cannot do this, because people have to go about their business. Then, I believe, the best thing is plenty of good food and iodide of iron.

A NOTE ON THE DIAGNOSTIC VALUE OF X-RAY PHOTOGRAPHY IN OSTEO-ARTHRITIS.

By E. W. H. SHENTON, M.R.C.S., L.R.C.P.,
Radiographer to Guy's Hospital.

THE Röntgen-ray appearance of osteo-arthritis is sufficiently characteristic to make radiography a useful adjunct to other methods of examination. The distinguishing features of osteo-arthritis seen through an X-ray medium are threefold: a pleasing picture, a curious pencilling-in of the cancellous tissue suggestive of a skeleton leaf, and an undue transparency of the bone at a joint region. As in tubercular joint conditions a blurred ill-defined picture is the rule, so one expects in osteo-arthritis a brilliant picture and well-defined image. Two of these characteristics, the brilliancy and fine pencilling, are to be found in the photograph; the third, that of transparency at a joint, is observable upon the screen. Various joints in this patient showed this light area, and the effect was as if, in a picture, the joints had been partially rubbed out with indiarubber. This appearance has been of diagnostic use in the case of a patient with pain in the lower end of the radius from no evident cause, and where later the more usual signs of

Normal hand.



Osteo-arthritic hand.

FOUR CLINICAL LECTURES
ON
SOME AFFECTIONS OF THE KIDNEY.

Delivered at Westminster Hospital.

By A. H. TUBBY, M.S., F.R.C.S.

LECTURE I.

GENTLEMEN,—I propose to deliver four lectures on certain affections of the kidney. The first one will be devoted to the subject of movable and floating kidney especially, as we have cases of that affection in our wards.

The first case is that of a nurse, æt. 35, who complained of pain in the right side. The history was that since July, 1899, she had suffered from dull aching pains in her right side. Sometimes the pain was sharp and increased by deep breathing or exertion of any sort, and she says that a month ago she consulted a doctor and was ordered to bed and poulticed on the right side. After two weeks rest in bed the patient was able to resume her occupation. But after a time she became worse, and accordingly she was painted with iodine without any good result. She went to bed again for a couple of weeks, and was then able to get about once more. Since then she has relapsed twice, and the pain has become more severe. On admission the mobile kidney was readily felt.

In the second case the doctor was said to have diagnosed pleurisy, but two months ago he suggested floating kidney, and the patient therefore took steps to enter a hospital. About nine months since the patient suffered from conjunctivitis, but with that exception she has had good health. With regard to the family history, the father has had an attack of paralysis; the mother is quite healthy. The patient is a rather nervous individual and suffers very much from migraine. She has acute pain in her right side, and it extends towards the middle line, just beyond the umbilicus. The pain from which she is now suffering has lasted three days, during which the patient has vomited continuously. The urine which was passed during that time was very thick, full of urates, sp. gr. 1020, reaction acid. There was no albumin, blood, nor sugar in it. On examination the cause of the trouble was perfectly evident. There could be felt in the right hypochondriac region a kidney-shaped tumour. It was tender and gave rise on gentle pressure to a sickening pain, and when pressed it slipped away from under the fingers towards the vertebral column, downwards and backwards. It could not be pushed towards the iliac fossa, nor underneath the front ribs. From the history and the signs there was no doubt that it was a case of movable kidney. Then there were certain negative points: the absence of blood, of pus, and of tubercle bacilli in the urine. These were all important points in the diagnosis. It was decided to operate upon her, and accordingly an incision was made between the last rib and the crest of the ilium on the right side. The incision was five inches in length. The colon was first discovered, and afterwards the kidney, and the latter was brought up into the wound. Five kangaroo tendon stitches were put in, so that the kidney was firmly anchored to the abdominal parietes. In that particular case I closed the wound up by sutures through the whole length. Three days after that she complained of considerable pain about the incision, so I had to open the wound between a couple of stitches, and a small amount of pus came out. I think that as the kangaroo tendon stitches were pretty large, they must have allowed a little leakage of urine from the kidney substance, which had become decomposed. The patient was kept at rest, and on the whole did very well, but she still complained of rather odd pains, so she was sent to the seaside for a month, and improved. She has had considerable swelling of her feet since the operation, and I do not know the cause of it. She is anæmic, but there is no albumin in her urine, and the swelling may be due to the anæmia. There is one possibility, viz. that in fixing the kidney the ureter became kinked, and so backward pressure was produced, and therefore the œdema. I believe such cases have occurred where it was found necessary to cut down upon the kidneys again and release the attachments.

The next case was a very straight-forward one. It was that of Edith M—, æt. 20. She was admitted on May 4th, and discharged June 8th of last year. Four years ago she fell on her right side on the bedstead, and then she had had dull aching pain in the right side, off and on. There had not been hæmaturia at any time, but she said she had

Digitized by Google

passed rather a smaller quantity of water than usual. Six months before admission the condition had been worse, and she could not get about on account of aching pains. Her menses were regular, but she had had more pain at her periods since the accident, and for the last twelve months she had worn an abdominal belt. In her case the signs were evident. There was a renal-shaped tumour on the right side, which slipped about, and pressure on it gave rise to a certain amount of sickness. It maintained an equable size and could be pressed back into the lumbar region; it could not be pressed towards the umbilicus nor beneath the ribs, and the renal dulness in the loin was absent from time to time. The kidney was cut down upon, and sutured by seven kangaroo tendon stitches. Having learnt from the previous case, I had a drainage-tube put in, which Mr. Salter removed some days afterwards.

We will now pass to a general description of the affection. The first point in dealing with the kidney is, roughly, to remember its anatomy. You are well acquainted with the size of the kidney, and you will remember its position. If lines be drawn upwards through the long axis of the kidneys, those lines, if continued, will meet at a point about the middle of the sternum. That shows that the upper ends of the kidneys are approximated towards the middle line, and they are also pushed somewhat forwards. It is important to remember the position of the hilum of the kidney, and it is situated just opposite the first lumbar vertebra. The right kidney has different surroundings to the left. It is in contact with the liver, a point I want you to bear carefully in mind, because there is no doubt that contact with the liver has a good deal of influence on the recurrence of movable kidney on the right side. It is also in relation to the duodenum and hepatic flexure of the colon, whereas the left kidney touches the fundus of the spleen and pancreas and the beginning of the descending colon. The relationship of the colon to the kidney is somewhat important. It is curious that if you have any disturbance of the kidney at all, either stone, movable kidney, or hydronephrosis, you almost always hear of certain colic disturbances, these taking the form of flatulence, colicky pain, and constipation. It is difficult to know why that is so, and it is vague to say it is a nervous affection. We want something more definite than that. When the right kidney is enlarged the ascending colon is always found in front of it, and you can easily determine that fact by percussion. If you percuss from the erector spinæ to the front, you will find, first of all, renal dulness, and outside that colic resonance. That is an important matter to bear in mind. When the left kidney is enlarged, you have not only the descending colon in front of it, but also a small part of the transverse colon as well. That is to say, the colon passes vertically up in front of the right kidney, and on the left side it loops over in front of the left kidney in the form of a semilune. There is one important point to remember in operating upon the kidney, viz. if you pass from the vertebra towards the hilum of the kidney you are in contact with the peritoneum, and that is the portion of the peritoneum in which the large meso-colic vessels are found. It is of the utmost importance that these vessels should not be damaged; otherwise gangrene of the colon may supervene. With regard to the position of the structures about the hilum of the kidney, the anatomy books are not quite correct on that point. There is often a good deal of modification; for instance, the vein is not always clearly in front of the artery. You will find the vein split up into a number of branches, and these branches all surround the artery. You should remember that the kidney lies in a bed of fatty tissue. There is no doubt that the perinephritic fat is the suspensory ligament of the kidney. Another point is, how is the kidney kept in position? That is a pertinent question, because you must remember that the state of the kidney is very different in the living from the dead subject. In the living body it is a large pulsating organ, which is liable to shock, and from a blow may become dislocated out of its bed, so giving rise to movable kidney. How then is it that the kidney can be kept in its place during the movements of the body? The first means to this end is undoubtedly the perinephritic fat, the kidney being well enveloped in it.

Another way by which fixation of the kidney is effected is what I may call the close adaptation of the viscera to the kidneys; it is the pressure of the viscera in the immediate neighbourhood which keeps it firm. That is proved in this way: if you look at the anterior surface of the kidney in a frozen specimen you will find that the kidney

Digitized by Google

shows numerous depressions and elevations. On the right side there is a distinct impression from the liver. There are also slight colic and duodenal depressions. On the left side there are distinct impressions from the spleen and pancreas, and it is the reciprocal pressure of one abdominal viscus upon another that keeps them in their place. I do not think it is sufficiently appreciated that in the living body there is no space in the abdominal cavity at all; there is really no true peritoneal cavity, but a very close packing. One usually imagines that the intestines are round, but that is incorrect. There is no doubt the intestines, as they lie side by side, are four- or six-sided tubes rather than circular, on account of the close packing of which I have spoken.

Another important point to bear in mind in the anatomy is the relation of the kidney to the pleura. The pleura comes down to the twelfth rib, and it is quite possible to make a mistake as to which is the twelfth rib. There is a case recorded in which there were only eleven ribs present, and when an incision was made on to the kidney the pleura was opened; the case happening to go septic, the patient died. It is therefore important to count the ribs before operation. And there is another fallacy to be borne in mind, namely, that the last rib may be so short that you may miss it altogether, and you may therefore count the eleventh rib as the twelfth.

Between movable and floating kidney, there is a clear distinction. In the case of movable kidney the organ moves entirely subperitoneally, and the movements are in the perinephritic tissue. In floating kidney, on the other hand, the movements are intra-abdominal, not in the fat, but absolutely intra-abdominal. For the kidney to be so mobile it must have a peritoneal pedicle or meso-nephron.

Movable kidney is common; floating kidney is very rare. In floating kidney the amount of mobility varies. There is no doubt a certain amount of normal mobility, that is to say about one to one and a half inches, especially in the case of women who have borne children; and the question arises, in what way does a movable kidney travel? Firstly, it may move about in atrophied perinephritic tissue. Secondly, the kidney and the surrounding fat may move in one mass behind the peritoneum. Thirdly, the kidney may move in the fat, and the fat may itself move behind the peritoneum. The amount of mobility varies considerably; it may be from one and a half to two inches, or even more. The exact extent to which the kidney may be disturbed depends very much on the length of the renal vessels.

As to the condition of movable kidney, it is often a healthy one. It is occasionally found diseased, and some of the cases in which it has been so found which have been recorded are as follows. There was a case in which it was found to be carcinomatous, another tubercular, and there are yet other cases of hydronephrotic kidneys. But in these cases the hydronephrosis is almost always secondary. As Mr. Clement Lucas has put it, in these cases the kidney brings about its own destruction; it moves and twists and blocks the ureter, so that the fluid accumulates in the pelvis, and the kidney becomes dilated and enlarged. In some of these cases you have pyelitis, as Dr. Dickinson pointed out.

Floating kidney may be a congenital condition, and in that case the kidney has a distinct mesentery; yet it is quite possible to imagine that if a movable kidney were allowed to persist it would gradually drag on its vessels and on the peritoneum, and so become a floating kidney.

As to the ætiology of movable kidney, it is six or seven times more common in women than in men: and why? I think the reason is dependent upon the parturient functions. And secondly, you often notice in some women a rapid absorption of fat throughout the body, and no doubt the perinephritic tissue shares in it. A third cause is traumatism. You will remember a case I narrated a few minutes ago of a girl who fell across the bed, and so started the kidney out of its position. In the fourth place, the right kidney is four times more frequently movable than the left. That may be due to the presence of the liver on that side, and also to the prevalence of tight lacing. Tight lacing is very apt to push the kidney out of its bed. In a post-mortem which I saw at Guy's Hospital some years ago, a woman who had tight-laced had her liver nicely arranged in two parts, an upper part, and then a band of fibrous tissue, and then a lower part in the iliac fossa. After parturition there is often a loss of flesh, and there may be a considerable laxity of the parietes. Another point is that the kidney of the pregnant

Digitized by Google

woman is of increased weight, or it may be large on account of tumour or tubercular disease. Greig Smith noticed certain anatomical conditions in cases of mobile kidneys, namely, long flexible spines, sloping lower ribs, especially in the upper classes, who ride, play tennis, etc., and attempt to lead a masculine life.

Symptoms.—I wish you to remember that in many cases the patient is neurotic, and if you speak of a movable kidney to her the idea becomes fixed in the patient's mind. There may be the usual movements consonant with health, but the patient will interpret these into pathological conditions, and will be unable to get movable kidney out of her mind. The same applies to a good number of men; they would be well if one had not happened to have mentioned their kidney. In other people you may feel their kidney moving considerably, but they know nothing of it. In a certain number of cases the neurosis of the patient is fixed upon the kidney. A few of these cases, especially in movable kidney, really start with pain, and from the worrying effects of this they become neurotic. I want you to bear this in mind, because it is lack of giving due weight to it which leads to the treatment of these conditions coming into disrepute. A large number of so-called movable kidneys should not be operated upon at all, because the patient is merely neurotic. But a small number of cases require operating upon, because the effects are a serious disablement in life.

The symptoms are pain, which is constant in the loin. It is dull, aching, and dragging, and is increased by movement. The pain shoots down the abdomen and towards the thigh. It is increased by the presence of constipation in the patient, and it is occasionally paroxysmal, and may resemble renal colic without the blood. Vomiting is fairly constant, with gastric disturbance, frequency of micturition and tenesmus.

The actual signs are best marked in floating kidney, because the conditions are more aggravated. There is a kidney-shaped tumour, which you can feel, but I should advise you not to attempt to do so too much, because I have been assured that the pain of squeezing a kidney is very much like that caused by squeezing the testicle. This tumour is tender and painful, and it slips away from under the hand towards the loin. That is the most typical sign of all. If the kidney is very mobile there is no renal dulness in the loin at all, and in place of it you have a general lumbar resonance. You can often produce this symptom by turning the patient on his face, and then the kidney will fall forwards, and you will lose the renal dulness. A kidney which is floating may be intermittently enlarged from temporary hydronephrosis.

The diagnosis should be made from distended gall-bladder. You should take note that in movable right kidney there is occasional jaundice found, so that the diagnosis is somewhat complicated. As a rule the gall-bladder is superficial, but not always. The gall-bladder may be traced up very often under the edge of an enlarged liver, whereas a movable kidney slips away under the edge. A gall-bladder can be traced continuously up; a movable kidney slips away. Growths of the pylorus have been mistaken for movable kidney, but they ought not to be as a rule, because with growths of pylorus there is a definite train of constant symptoms, whether the tumour moves or not; there is vomiting irrespective of meals and of movement, which is not the case with movable kidney. Growths in the intestine have been mistaken for movable kidney, and so have impacted fæces. The latter may be diagnosed by putting one's hands firmly over the mass and pressing the tips of the fingers in, when you may get a doughy sensation.

The *treatment* should be either (1) operative or (2) palliative. The relationship between the two is this; that no patient should be operated upon until the palliative treatment has been tried and failed. That is all-important. I think the great risk of surgery at the present day is excessive operating. I advocate that in all cases you should first try palliative treatment, and only after this operate. The palliative treatment consists in the adjustment of a properly fitting abdominal belt, which should have a trial of two or three months before operation is undertaken. If the abdominal belt is made properly, it succeeds in a large number of cases; if it is made improperly it fails altogether.

When should the operation be undertaken? When should you take the responsibility of advising the patient to have an operation on his kidney? (1) If you fail to hold a movable kidney in position

Digitized by Google

by a belt; (2) if the pain is such that it cripples the patient's life; (3) if the pain causes excessive vomiting; (4) if the kidney is jammed between the ribs and the iliac crest, which sometimes happens, and patients afflicted in that way have excruciating pain in the region of the kidney, with vomiting and collapse; (5) if there is hydronephrosis present. The operation is called nephrorrhaphy. You make an incision half an inch below the last rib, commencing at the outer border of the erector spinæ, and passing outwards four or five inches through the posterior margin of the external oblique, and cutting the internal oblique, transversalis, and lumbar fascia, and then you look for the posterior part of the transversalis fascia. Cut through that, and open the perinephritic tissue. There you will find the kidney. The more you keep towards the middle line the less likely you are to damage the colon, or even to see it.

With regard to fixation of the kidney, that is a matter of considerable difficulty. When this operation was first undertaken it was the custom to put in one or two sutures in the kidney to keep it in position. Now, however, it is the usual procedure to put in seven or eight. The kidney substance is so friable that there is a distinct danger of the sutures tearing out. There is an operation which is recommended which I have tried. It is this: to stitch the capsule of the kidney to the lumbar fascia. The capsule over the convexity is split, and then the flaps are carefully stitched to the lumbar fascia as closely as possible. It is claimed that it gives excellent results. I have done it by a method nearly similar, that is to say, I have split the capsule transversely, and stitched the edges to the fascia. But I have not seen much better results than by stitching through the kidney substance. The operation takes a long time to do, and, of course, must be correspondingly exhausting to the patient. The operation of nephrectomy is a last resort. Only when the kidney which is movable is the seat of carcinoma, or the seat of tubercle or degenerations from stone, or if nephrorrhapy has failed, and the patient's life is unbearable, should we resort to nephrectomy.

By an unfortunate accident the usual acknowledgment for the illustration No. 2 on page 66 in last week's issue due to Mr. Bland-Sutton did not appear.

THE PRACTICE OF BLOOD-LETTING.

By J. F. BRISCOE, M.R.C.S.

(Continued from p. 80).

The next cases that I wish to bring to your notice are those which result from a poisonous dose of the vapours and gases, some of which are used as anæsthetics and some as illuminants. Chloroform, ether, nitrous oxide gas, and combinations of these are examples of the former. All these aëriform elastic fluids if inhaled beyond a certain point may cause asphyxia, either through the nervous system or the red corpuscles of the blood.

As I have said before, if the heart were exposed to view during asphyxia we should frequently discern the distension of the right cavities. I refer particularly to true asphyxia from deprivation of atmospheric air, rather than to the asphyxia of chloroform or prussic acid, where there is another element of danger thrown in: namely, the properties of the individual members of the neurotic poisons, which would seem to act on the nerve cells themselves. It is always desirable in making a *post-mortem* on anyone who has died from a poisonous vapour, an anæsthetic, in true asphyxia, or convulsions, to make the examination at once. In an 'Essay on Asphyxia,' Dr. G. Johnson, quoting from Harvey's 'Second Disquisition on the Circulation of the Blood,' says, "I have several times opened the breast and pericardium of a man within two hours after his execution by hanging, and before the colour had totally left the face, and in presence of many witnesses have demonstrated the right auricle and the lungs distended with blood, the auricle in particular being as large as a large man's fist, and so full of blood that it looked as if it would burst. This great distension, however, had disappeared the next day, the body having stiffened and become cold, and the blood having made its way through various channels."

Dr. Alfred Swaine Taylor in 'Principles and Practice of Medical Jurisprudence,' and again, Drs. Guy and Ferrier, confirm the views of Dr. G. Johnson with regard to the physiology of asphyxia. However, Dr. Sanderson somewhat disagrees with Harvey. Again, Drs. Michael Foster, Gerald Yeo, and Landois and Stirling

Digitized by Google

hold opinions which are in accord with Dr. G. Johnson; but they say that, owing to the force of the rigor mortis of the left ventricle, and the greater capacity of the systemic veins, the left side is found comparatively empty some time after death, and at post-mortem examinations the right side alone is found overfilled. Dr. G. Johnson in his 'Essay on Asphyxia' clearly gives an instance, page 14, to prove that the left cavities of the heart are not emptied by rigor mortis. The whole essay is worthy of the student's perusal. In making a special examination of the heart at an autopsy, it is desirable to dissect very carefully, and avoid wounding a vein. Otherwise it is obvious that a cavity of the heart could be easily emptied, and thus an erroneous report of the case chronicled. I would suggest in all post-mortem examinations where the pathologist's inquiries are directed to the heart, that the assistant should be instructed to place firm ligatures about two inches above the large vessels which enter its cavities. The organ can be thus removed intact, and doubt as to the amount of blood in any of the cavities will be avoided. Before closing this paragraph I should like to refer to an experiment by Dr. Massey, late of Nottingham, which has been repeated by others. He affixed a ligature on the trachea of a dog of 19½ lbs., and when convulsive movements had ceased, he rapidly opened the chest and found extreme distension of the right cavities of the heart, while the left cavities were completely empty and flaccid.

I will take nitrous oxide as an example of the vapours or anæsthetics which produce asphyxia. During its administration we see the signs of an epileptic fit, but directly the mouthpiece is removed the phenomena pass off. There are undoubtedly persons who have been violently convulsed during the administration of nitrous oxide. At a meeting of the Society of Anæsthetists, March 17th, 1899, a case which ended fatally after the administration of gas and oxygen and chloroform was recorded. The anæsthetists gave the ordinary restoratives for two hours, with artificial respiration. It was reported that at the autopsy nothing was abnormal and everything was healthy, and yet it was stated that the right ventricle was dilated. Surely this youth's life, he was 17, might have been saved by venesection. But Mr. Granville is to be thanked for publishing the case, and I feel sure if many more of our failures were recorded, we should be all the wiser, and become possibly more expert. I myself once had a fatal case while administering ether. The patient, a bloated woman, and thus an unpleasant case to anæsthetise, was undergoing an operation on the breast. No post-mortem was allowed, but I feel sure she was suffocated, and ought to have been bled. Artificial respiration and the usual restoratives were employed without any success. But artificial respiration is not to be always despised; there is, for instance, the medical man who restored the life of an infant after fourteen and a half hours artificial respiration. The case is reported by Dr. G. E. Keith, 'Lancet,' November 24th, 1900, but no pathological explanation is satisfactory as to the origin of this anomalous affection of the respiratory centre, which needed such artificial procedure in a new-born child, unless it was toxic or imperfect vitalization owing to some unexplained cause. The question of venesection might have been a point in the case, but I very much doubt under the circumstances, in such a tender subject, whether the escape from death would have been accomplished by this means. The patience and the resource of this skilful practitioner was beyond all praise, and it is to be hoped he was rewarded for his heroism. The result of his treatment proved beyond a doubt his practical skill in the treatment of impending death. However, such saving of life is worthy of the highest respect from the profession, and it is a point in favour of adopting treatment according to the peculiarities and merits of the case, and not being too routine in practice. In the case of a bad subject, I believe that anæsthetics may cause death from paralysis of the heart, beginning with distinct respiratory changes, and terminating in distension and paralysis of the right heart. However, there is no definite ruling at present with regard to the pathology of the lethal effects of the anæsthetic vapours. So that in considering venesection for anæsthetic toxæmia we approach a very difficult question. Specialists have an extremely low percentage of deaths under chloroform, and practically none under some anæsthetics. They have also the worst cases, and deal with them both in hospital and private. But, as Dr. Dudley Buxton says, "if the anæsthetic is given in excessive quantities, leading to gradual and general accumulation in the whole

Digitized by Google

blood-stream, venesection is of great value." This authority recommends with blood-letting intracellular transfusion. If the advice of the 'Lancet,' October 27th, 1900, "Anæsthetics and the Medical Student," tends to increased practical education, perhaps we shall also hear that "blood-letting" is likely to receive its share of consideration. If venesection is to save the life of one poisoned by an anæsthetic, it must be done first, and not as a last resource. Hoping not to be thought too discursive, I should like to remind my readers that artificial respiration may be practised in too routine a way. For instance, Dr. Dudley Buxton, "The Management of the Anæsthetic in Cases in which Respiratory Impediment Exists," 'Lancet,' June 16th, 1900, points out, "while speaking of perils occurring during anæsthetising in thoracic operations, I would mention the danger in these cases of performing Silvester's method of artificial respiration, should breathing fail. In a number of instances published in the journals in which this restorative measure was recorded as having been adopted, a fatal conclusion to the case occurred." Mr. Bryant's practice is worthy of mention, for all the time I was his house surgeon I never saw a death from an anæsthetic. He used to say, "If I have a careless house surgeon I always have the A.C.E." Again he never desired the drug to be pushed. It is for the profession to decide whether there is an idiosyncracy in the subject who succumbs to an anæsthetic, whether an error has arisen in its administration, or whether the vapour is impure. If venesection is a remedy in uræmic toxæmia, surely it is so in anæsthetic accidents. In looking over one year's deaths from anæsthesia, 'Brit. Med. Journ.,' 1892, I see there was a death from chloroform at University College Hospital in which venesection was performed on the external jugular; but the house surgeon says, "At no time after the first attack was the heart felt, or was there any radial pulse." Had perhaps the venesection been done before the artificial respiration, etc., there might not have been a post-mortem revealing, as it did, distension of the right cavities of the heart with fluid blood, while the left side of the heart was empty. Sir Lauder Brunton remarks on "Death during Chloroform Anæsthesia," Annual Meeting Brit. Med. Assoc., 1891, "that we may say that the great risk of death from the action of chloroform lies in the occurrence of asphyxia during its administration." This death, commencing at the lungs, arises, we suppose, from toxæmia of the respiratory centre, and claims our due attention. Dr. John W. Ogle quotes, *vide* 'Syd. Soc. Trans.,' vol. i, Dryden, the poet, who was contemporary with Sydenham, and who uses the expression "to breathe a vein"—

> "The ready cure to cool the raging pain
> Is underneath the foot to breathe a vein."
> Georgics iii, 460.

But in these days he must be a bold and courageous anæsthetist who steps in with the lancet for impending death from anæsthesia. It is, as Dr. Duffin says in his article ('King's College Hospital Reports,' vol. i), "The Abstraction of Blood clinically considered," "that a useful remedy has fallen into disrepute owing to the want of discrimination in cases of this kind."

Some little time back I was watching, in a London hospital, an operation on the skull for some supposed cerebral growth. The subject was a plethoric-looking man of fifty years. The pulse was getting small, the breathing hurried, and the face blue. The anæsthetist was alarmed. The circle of bone was just removed, and the gouge was being used. The vessels over the dura mater were fully congested. The "sister" was ready with restoratives, and preparations were made for artificial respiration. But someone said, "open a vein." Two veins were incised at the trephine wound, and it was most gratifying to watch the colour return to the man's face, and the breathing settle down to normal. Of course we are prepared to hear that recovery was due to the removal of the inhaler. There are some subjects so susceptible, owing to idiosyncrasy, that the smell of certain flowers, or the hayfield, will produce distressing symptoms; and with drugs, and not least with anæsthetics, there can be no doubt of similar idiosyncrasies. The vital capacity of the chest is likewise to be considered, or, as Dr. Dudley Buxton would say, the "personal respiratory equation of the patient," the strength of the heart, the nervous organisation, and above all, the previous life of the patient. There are some of us with weak hearts, and others with weak digestions, and yet when we examine the organs we can find nothing amiss. The infrequent pulse of 40 or 50 beats to the minute

Digitized by Google

is not supposed to interfere with longevity. Yet, whether the bradycardia be of a congenital or acquired nature, it behoves us to be circumspect when we administer the anæsthetic vapours to these persons. As with the bleeder, who easily succumbs from the slightest wound, so a fatal result from anæsthesia is possible with an habitually slow pulse. It is generally believed that if the organs of the body are healthy, the patient has the best chance of recovery from a shock, an operation, or a serious illness, but of all the organs good kidneys seem most important for the patients of operating surgeons. Atonic heart disease is as good a phrase as atonic dyspepsia; and it is in such cases that we should be cautious when we administer anæsthetics. If we define a poison as a substance which, having entered our system, is capable of destroying life, surely the anæsthetics come under this heading. Carbonic acid, carbonic oxide, coal gas, and many others, cause death by asphyxia, and thus their effects may be met by blood-letting. The nature of the changes gas poisoning produces is unknown apart from their manifestations, according to Dr. Lorrain Smith, who writes on the pathology of gas poisoning, illustrated by five recent cases, in the 'British Medical Journal,' April 1st, 1899. He states that the patient poisoned by coal gas is suffering from a form of suffocation or asphyxia. The great medical jurist, the late Dr. Alfred Swaine Taylor says "that all gaseous poisonous vapours produce death from asphyxia. In the case recorded of the diver, the pathology will compare with those cases of poisoning by carbonic oxide at the Snaefell mining disaster which occurred two years ago. There is a Blue-book on the nature of the disaster, beside Professor Le Neve Foster's official report, with a special report by Dr. Miller, of Laxey. An account of these thirty-one cases of CO-poisoning will be seen in the 'British Medical Journal,' July 2nd, 1898; nineteen were found dead, and eleven reached the surface alive. It would seem that the phenomena of CO-poisoning are similar to the effects of nitrous oxide in the first stage, for there is quickened and hurried breathing. To sum this up briefly, it is stated in Dr. Miller's report that in acute asphyxia by CO the respirations stop before the heart fails. In reading the report it struck me very forcibly that in treating cases of CO-poisoning venesection should play an important part. The five cases of Dr. J. Lorrain Smith are equally interesting, from the fact that at the autopsies there were found extensive hæmorrhages into the tissues, and also congestion of the organs. These are in accordance with the case of the diver on whom I performed blood-letting, and whose life was saved by this means. Before I conclude this interesting subject of gaseous poisoning, I can hardly pass over two recent cases of poisoning by carbon monoxide, reported by Dr. C. R. Elwood (*vide* 'Lancet,' September 15th, 1900). Two women in a workhouse, aged respectively fifty-eight and thirty-one years, were poisoned by the fumes from a stove. The elder one died, and these are the words which Dr. Elgood gives to us in the 'Lancet': "She was not breathing, there was no pulse, and no cardiac sounds could be heard with the stethoscope. There was, however, a good colour in her lips and face; so much was this the case that, in spite of the fact that the woman had not been observed to breathe since she was found one hour before, artificial respiration was resorted to, and was kept up some considerable time, until its obvious uselessness was apparent to all, for she never gave a gasp." It would seem that the doctor was deceived by the good colour of the lips and face, for the blood at the post-mortem, was found by Dr. Elgood to be brighter than normal, and all the organs were of a brighter colour than usual. Dr. Elgood states, "the corpse was the occasion of a good deal of uneasiness to the nurse who had to 'lay it out,' for from the appearance of the body she could not bring herself to believe that the woman was dead." The post-mortem was made about forty hours after death. Now I contend this patient might have been bled. To be critical, the case was one of CO-poisoning described in the text-books on forensic medicine, the post-mortem signs being redness of the face, with reddish patches on different parts of the body, and the characteristic cherry-red stable compound. The treatment consists, according to the text-books, in removal to fresh air, artificial respiration, venesection, and transfusion of arterialised defibrinated blood.

Later on, in Dr. Elgood's paper, he states "One would imagine from *a priori* reasoning that, as carbonic oxide forms such a stable compound with hæmoglobin, there is but little chance of setting free this combined hæmoglobin, and that

Digitized by Google

the only method of treatment would be to remove some of it and inject fresh into the system. And this experience fully proves to be the case. A case is related where a man was saved by this means, after being in an apparently hopeless state for forty-eight hours. He was bled to 800 grammes, and 110 grammes of defibrinated blood were injected." I am sure the profession is indebted to Dr. Elgood for publishing these two cases and giving us his views.

Dr. Lorrain Smith, in his paper on "Gas Poisoning," remarks, "when a person exposed to CO is found while still breathing and placed in normal air, the CO is gradually eliminated from the blood, so that after some hours it has completely disappeared." But in his paper he records a case of gas poisoning treated by Dr. T. R. Davison, of Belfast, in which the patient died after four days from pneumonia, and never recovered consciousness. Here is a case, perhaps, in which extravasation of blood was a pathological sequence, and in which I contend venesection was indicated.

Before closing the subject of gaseous poisoning, I will allude to another instance where I think venesection was indicated. The case was recorded in the 'Lancet,' March 31st, 1900, by Dr. Scott Thomson, of Rhymney Works, Monmouthshire, under the title "Death from Carbonic Dioxide Poisoning in a Brewery." The heart had, according to the report, ceased to pulsate; there were no visible respiratory movements, and there was some frothy fluid about the mouth and the nostrils. The skin was pale, the lips and eyelids were slightly cyanosed, the eyes were half open, the pupils widely dilated, and the expression of the face was calm. The doctor arrived at this stage, but surely the subject had succumbed to paralysis of the heart. Is it to be wondered at that he failed to resuscitate the patient by the Silvester method of artificial respiration, although he practised it for forty minutes?

At the Annual Meeting of the British Medical Association, 1899, Dr. Ewart, in the Section of Medicine, described a novel method of administering carbonic acid gas in dyspnœa and anginal pain, and he extended this line of treatment to congestive affections of the lungs; in fact he implied that it was useful also in heart affections. After his paper was read I expressed some surprise at the deductions which he had drawn from his experiments. I pointed out that the very condition for which this novel treatment was advocated was associated with an excessive amount of carbonic acid gas in the blood, and would be better treated by a moderate bleeding, which is always recommended by those who know anything of the practice of blood-letting. I cannot believe that the inhalation of carbonic dioxide is likely to find a permanent place in cardiac therapeutics. We are bound to respect the opinions of other people, and not least that of Dr. Ewart, and my criticism requires the support of others more qualified to express an opinion. But, as I have before said, in "Conditions Indicating Blood-letting," is it not a fact that the few therapeutic agents which exert power specifically are not more in number than the fingers of our two hands? The theory of medicine is one thing, and the practise another. It is very easy to give a patient a dose of carbonic acid gas, for, as Dr. Barr says, "you can pinch his trachea." But, instead of administering carbonic dioxide in heart and lung affections, I should at once advocate venesection when you find the venous system engorged in primary affections of the lungs, or in secondary conditions, as in heart disease. This is no novel agent, on the contrary it is as old as the hills.

Modern text-books tell us of the importance of venesection when suffocation is impending, as in capillary bronchitis. Again, in pneumonia, and in all cases of great dyspnœa and threatened cardiac failure, life may be saved by a timely bleeding. Dr. Eustace Smith recommends it in children where there is threatened failure of the heart. Again, at Great Ormond Street Children's Hospital venesection is performed, and Dr. David Lees, physician to this institution, considers the removal of blood a most valuable therapeutic measure, and he practises it at Great Ormond Street, (*a*) in most cases of failure of compensation in heart disease, especially with right heart failure as indicated by increased heart dulness, which may be easily obtained by careful light percussion in the fourth intercostal space, (*b*) in pneumonia and capillary bronchitis with increase of the cardiac dulness to the right and the evidence of right heart failure. We read in Fagge and Pye-Smith's work on 'Medicine' that some fatal cases of pneumonia might be saved had we but the courage to

Digitized by Google

relieve the labouring heart by judicious removal of blood. Although admitting that the greatest danger in pneumonia is failure of the heart, I cannot believe that this is not due to pulmonary obstruction. On the contrary, if it is not the fever, what then is it that produces this failure? Undoubtedly the very same thing which occurs as in all asphyxial affections, namely paralysis of the heart's action from distension of the right cavities of the organ. The fact that pneumonia has been treated by infinitesimal doses of useless drugs is no criterion as to the value of blood-letting. It would seem that all diseases of a more or less serious nature may cure themselves. Yet there are states and conditions arising in all maladies which will determine the life of the subject. This is encouraged, moreover, by the inherent instability or weakness of the vitality of the cellular life of the individual. The inability of the remaining healthy portion of a diseased organ to maintain the function of that organ is a direct cause of death; when, on the other hand, if the unaffected tissue is of good quality it defends itself until it is relieved. Under these circumstances heroic measures, such as blood-letting, are sometimes indispensable to save the patient from a perilous position. What really do we know of the true action of any drug when a vital organ is in jeopardy? The result is often problematical when we apply our drugs, and time is an essential element of success, for a moment's delay may involve the loss of a life. The same intelligent reasoning is just as necessary in blood-letting as when we deal with remedies like arsenic, belladonna, strychnine, etc. The host of new remedies and methods of treatment which periodically change into another fashion owing to their uselessness, the readiness with which practitioners will thrust these remedies into the stomach, under the skin or into the rectum, and the doubtful therapeutic value of most of the manufacturing chemist's compounds, is bewildering enough to any practitioner.

To recapitulate, the indication of venesection is mechanical impediment to the cardiac or respiratory movements. We see it in the livid look, the oppressed breathing, the anxious expression, the sitting-up posture, the œdematous tissues, and above all the weak small pulse. Terrible as it may seem to recommend bleeding, yet over and over again the bold and thoughtful practitioner may alter the whole state of affairs. The relief from venesection is given long before one anticipates syncope; it is given while the blood is escaping from the vein. Surely these cases of distressing asphyxia, when perhaps a large extent of the pulmonary aërating surface is rendered useless, or again in heart affections where the cavities are distended, are beyond the reach of drugs of the pharmacopœa. Stimulation by brandy, strong beef teas, etc., is but a temporary stimulation, but if blood-letting is wisely practised the choking of the tissues, the suffocation, the paralysis, and the death of the subject may be averted.

Dr. Sansom, in a clinical lecture on the "Rheumatic Heart" (see 'Clinical Journal,' January 31st, 1900) points out very clearly the object and value of blood-letting in valvular disease of the heart. He says, "Then you act disproportionately if you give digitalis for any length of time, you act disproportionately upon the right ventricle. The more you get the right ventricle to work, the more you may drive the blood into the venous circulation, including the liver; therefore it is not likely to do so much good. Five- to ten-minim doses of the tincture of digitalis, or drachm doses of the leaves three times a day, do good for a limited time. When you find it does not do very much good you have to wait. In some cases it does not do good at all. The great difficulty is that the right ventricle is overdone. You may do a great deal then by letting out a little of the distension. Bleed from a vein, and learn to bleed; you may save lives entirely by bleeding properly, and at the right time. Some people will not hear of being bled, but they do not mind a few leeches. Dry cupping you can do, but wet cupping makes people flee from it in horror. Do not be afraid of popular prejudice. When you have thus relieved the tension in the right heart, digitalis will perhaps do good; but you can give another cardiac tonic, namely, convallaria and caffeine, but particularly convallaria."

The following are illustrative cases of blood-letting in chest affections:—

A. B—, female, æt. 30, suffering from severe double pneumonia, respiration very hurried, an anxious expression of countenance, lips blue, and every indication of asphyxia with a small pulse. Venesection was performed, the patient rapidly

Digitized by Google

improved, and a large amount of blood was removed.

J. C—, æt. 48, a large-built man, suffering with chronic disease of the valves of the heart. Legs œdematous, abdomen dropsical, much oppression of his breathing, and effusion into pleural cavities. The ordinary diuretics, diaphoretics, and purgatives failed to relieve him. He was bled, but not by the ordinary medical attendant, since he had never done the operation. Thirty ounces of blood was removed, and the relief was unmistakeable—it was immense and immediate. He was up the next day, and made a complete temporary recovery from the dropsical attack in question.

R. P—, æt. 75, male, temperate; two ribs were broken after a fall from a cart. He suffered from much pain in the chest; dyspnœa and facial anxiety was early noticed. Ordinary remedies failed to relieve him, and he was bled. Relief was immediate and permanent.

L. B—, æt. 55, male, broken ribs; symptoms became alarming on the third day. Patient was bolstered up in bed, his legs swollen, lips blue and congested, and the doctor in attendance considered him dying. A consultant was called in, and after thirty ounces had left the venous system the patient resumed the horizontal attitude. Asphyxial symptoms passed off, and from the point of dissolution the patient gradually improved and recovered.

In a patient suffering from a severe attack of pneumonia, with high temperature, cyanosis, rapid pulse and respiration bleeding to twenty-six ounces proved of marked benefit. A second relapse was treated by leeching, which caused five ounces to flow, and this did good, but, when bad symptoms returned for a third time, on the removal of twelve ounces of blood she rallied and recovered.

MR. JAMES BERRY ON DYSPNŒA CAUSED BY GOITRE.

The subject of dyspnœa caused by goitre is treated by Mr. James Berry in a very interesting manner in his valuable work on 'Diseases of the Thyroid Gland and their Surgical Treatment,' just published by Messrs. J. and A. Churchill. Mr Berry considers that of the various symptoms which may be produced by an enlargement of the thyroid gland, dyspnœa is, undoubtedly, by far the most important. In 95 out of the 126 cases in which he has had to remove a goitre the chief reason for the operation was dyspnœa. In 16 of the other cases some dyspnœa was also present, although it was not the main reason for the operation. Frequently, dyspnœa is the only inconvenience of which a goitrous patient complains, and for which he seeks advice. Often it is present to such an extent that a constant dread of impending suffocation hangs over the unfortunate sufferer, and prevents him from following any regular occupation. Only too often is death itself the result of the gradually or suddenly increasing dyspnœa. It is not very uncommon for a patient to seek advice with regard to dyspnœa, the cause of which is unknown until an examination of the neck reveals the presence of a goitre which had hitherto not been noticed by the patient. Cases have come to Mr. Berry's knowledge in which dyspnœa has been supposed to be due to asthma and bronchitis, and treated accordingly for months or years, the true cause lying, wholly unsuspected, in an enlargement of the thyroid. Such mistakes are most likely to occur in cases of small and symmetrical, or deep-seated tumours, and these are especially liable to cause severe dyspnœa. Mr. Berry devotes some space to a consideration of the various modes in which dyspnœa may be caused by disease of the thyroid, and to the varieties of such diseases which are most liable to cause dyspnœa. He divides the various ways in which an enlarged thyroid may cause dyspnœa in the following manner:—(1) Direct pressure upon the trachea; this is by far the most common and important. Occasionally other conditions have some share in the production of the dyspnœa, either alone or more often in combination with the preceding. Such are: (2) Pressure upon or other irritation of the recurrent laryngeal nerves; (3) direct extension of growth into the interior of the trachea or larynx (very rare, except in cases of malignant disease, in which it is common); (4) rupture of a cyst or abscess into the air-passages; (5) sudden hæmorrhage into the gland or from it into surrounding tissues; (6) œdema of the glottis or of the mucous membrane of the trachea.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 449. WEDNESDAY, JUNE 5, 1901. Vol. XVIII. No. 7.

CONTENTS.

PAGE

*A Clinical Lecture on Movable Kidney. By W. ARBUTHNOT LANE, M.S. 97

*With Dr. Hale White in the Wards of Guy's Hospital (*concluded*) 104

*The Ophthalmoscopic Changes met with in Bright's Disease. By PERCY FLEMMING, F.R.C.S. 109

*A Case of Acute Glanders. By W. C. C. PAKES, D.P.H. 112

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE
ON
MOVABLE KIDNEY.

Delivered at Guy's Hospital, May 24th, 1901.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

WE will attempt to treat this subject in a more or less scientific manner, dealing in order with its causation, its symptoms, its complications, and its treatment.

In considering its causation, I will first call your attention to certain deviations from what is regarded as normal in the position of the kidneys, and of other of the abdominal viscera, which are present under varying conditions.

In the ordinary vigorous subject who uses the chest freely in respiration, when in the supine posture the kidney is placed high in the thorax, only a small proportion of the organ being below the last rib, where it moves upwards and downwards with the ascent and descent of the diaphragm.

The position of the kidney, as seen clearly through a long lumbar incision, is on both sides much higher than that described as normal in text-books on anatomy. In such a vigorous subject the thorax occupies habitually a position intermediate between that of complete inspiration and that of complete expiration, it being possible to take in an additional supply of air freely, or to expel it without depending on the action of the abdominal muscles. This can be determined accurately by measurements of the chest. If the "symmetrical erect attitude of rest" is assumed in which the chest is retained in a position of complete expiration, respiration being purely abdominal, the kidneys can be felt at a level which is distinctly lower than that observed in the recumbent posture.

There are, as you know, a very large number of people who perform their respiratory functions in a most imperfect manner, depending, under ordinary circumstances, for the interchange of gases entirely upon the action of the diaphragm and abdominal muscles. The chest is retained habitually in a

Digitized by Google

state of complete expiration, the trunk occupying the "symmetrical" or "asymmetrical position of rest." Figs. 7, 8, and 9 in 'Clinical Journal,' March 20th, 1901, represent such conditions in varying degrees of severity, in Figs. 7 and 8 they are comparatively slight, while Fig. 9 shows them in a somewhat exaggerated degree.

These people are able to increase the measurement of the chest to a small extent during forcible inspiration, after which effort the thorax returns at once to the normal state of the individual, which is that of complete thoracic expiration.

In consequence of, and in direct proportion to, the degree of imperfection in the manner in which the respiratory act is performed, the various functions of the body are carried out in a correspondingly unsatisfactory way.

This condition is seen in a much more marked degree in the female than in the male subject, for the reason that the male child escapes at an early age from the care of his mother and nurse, and so avoids much harm at their hands, while in the case of the female the physiology of the growing child is usually accommodated to that of the fully-developed and usually inactive female adult, with disastrous results to herself. That is why the thin, flat-chested, round-shouldered, stooping girl with the prominent abdomen is so conspicuous in our community. As she develops into maturity the dressmaker and the corset maker strive with one another to reduce her respiratory capacity still further, the former by stretching the materials composing the body of the dress tightly over the front of the flat thorax, rendering thoracic respiration impossible, and the latter, in her efforts to make the prominent abdomen as inconspicuous as she can, reduces the range of movement of the diaphragm to the smallest possible limits. By these means the kidneys are driven down still further from their original relationship to the large vessels of the abdomen. The corset under these circumstances exerts upon the lowly-placed kidney, especially during its descent in inspiration, just such a pressure as tends to force it still further downwards. The effect can be readily gauged by grasping firmly the loin of such a girl in the hand while she is in the erect posture, and directing her to take a deep breath. The kidney, which is easily held in the hand during the normal condition of habitual thoracic expiration, descends beyond it during the inspiratory effort, and remains beyond it till the grip of the encircling hand is removed, when it slips up again to its normal level. The constriction of the corset is not sufficient to effect this further displacement at once, though it gradually tends to produce it in the same manner as the grasp of the hand effects it while in action. At the same time, sufficient force acting on a single occasion in the position of the hand may produce a considerable permanent displacement of the organ.

It too frequently happens that at this period the young woman takes to expending more physical energy than her very limited supply of oxygen justifies her in doing, with the result that she becomes exhausted, and increases the extent of her resting position, and the consequent descent of the kidneys.

This occurs only too readily in whatever form of exercise she indulges in. I have already fully explained to you the mechanism of the shoulder girdle, with the mode in which force is transmitted from the arm to the spinal column, and have demonstrated how in the male the form of the chest is so arranged, that superior thoracic respiration is sacrificed to some extent to the efficiency in which force is transmitted from the upper extremity, and how in the female the thorax is constructed so that the respiratory process may be carried on as perfectly as possible by its upper part, the efficiency in which force is transmitted along the shoulder girdle being in consequence sacrificed very greatly. Also I showed you that as the chest approaches a position of complete inspiration, the more economically is such force transmitted; while, on the contrary, in the position of extreme expiration it is effected at a comparatively very great mechanical disadvantage, the dorsal spine being flexed still further. This is what happens when such a girl indulges in nursing or in any occupation or amusement involving the use of the arms. It is easy to see how it occurs in lifting a heavy weight, but it is not so marked or so obvious in their pleasures. For instance, in driving the ball in golf or in tennis, especially in certain positions, you readily recognise that great pressure must of necessity be exerted upon the kidneys, driving them downwards, and I have on several occasions known painful symptoms to originate in this manner.

What very few observers seem to recognise is

Digitized by Google

the close association between respiratory capacity and weight in the growing child. In my early attempts to treat the same physical conditions associated with the presence of the so-called adenoids by increasing the respiratory capacity, and by restoring to the naso-pharynx the mechanical factor upon which it depends for its development, I was very much struck by the increase in weight which accompanied the improvement in the respiratory capacity. To make this more clear I will give you two instances of the result of such simple treatment extending over two months.

On February 1st, 1891, N. B—, a boy, weighed 5 st. 13 lbs., and the tape, at the level of the nipple, measured 24½ inches in the normal position, which corresponded to that of complete expiration, and 27 inches on the deepest inspiration.

On February 15th he weighed 6 st. 4 lbs., and the measurements were 25 and 29¾ inches respectively. On February 28th his weight was 6 st. 7 lbs., and the measurements were 25 and 31 inches respectively. On March 30th he weighed 6 st. 10½ lbs,, and the measurements were 26 and 31 inches. In two months he gained a stone all but 2½ lbs., and his chest improved from 24½ and 27 inches to 26 and 31 inches.

His sister, E. B—, on February 1st weighed 7 st. 12 lbs., and measured 28½ inches in the normal position, also that of complete expiration, and 30 inches in the fullest possible inspiration. On February 28th she weighed 8 st. 7½ lbs., and measured 28½ and 34 inches respectively. On March 30th she weighed 8 st. 13 lbs., and measured 30 and 35¾ inches. In the two months she gained 1 st. 1½ lbs., and her chest improved from 28½ and 30 inches to 30 and 35 inches.

These results are clearly full of the greatest interest from a physiological as well as from a medical standpoint, and were obtained by making the child inspire and expire to the utmost while in the recumbent posture during certain periods of the day, so increasing the respiratory capacity. This simple exercise is performed in the recumbent posture in order to avoid any expenditure of energy whatever beyond that required to fill and empty the chest, and which would be necessary were the erect attitude assumed. Later, as the child accumulates energy, it may resort to the erect posture during the exercise for a change, expending a certain amount of it in retaining this position.

Even in the case of these very ill-developed girls nature makes a determined and powerful effort to stem the progress of physical depreciation, in that during pregnancy for a certain period the process of respiration is carried on almost entirely by the upper part of the thorax, whose capacity of freedom of action is very specially arranged for in its construction, and is fully developed during this period, often with the greatest advantage to the individual in the future. One constantly observes the immense permanent improvement in health, physique, and energy, which pregnancy may produce in the most feeble of girls.

Unfortunately, after a woman has ceased to be able to reproduce her species nature seems to take little further interest in her, and she returns rapidly to the condition of thoracic incapacity, with the flexion of the column necessarily associated with it, and loses a good deal of her original height. On this account elderly women usually avoid those garments which show their form, and favour looser apparel. In a young subject, such as I have described in the supine, and especially in the erect posture, the kidneys occupy a level which is very much lower than it is in the vigorous subject. In many cases in the easy erect or sedentary posture the kidney lies quite beyond the lower margin of the thorax, and the range of movement of the organ from the erect to the supine position is great. In a considerable number of these cases the patient suffers from pain in the loin, usually regarded as muscular, but which I have found to be generally renal in origin. Very often the kidney is tender on pressure when the erect posture has been occupied for a considerable time.

As time goes on, the kidney occupies a progressively lower level till it may fall down into the true pelvis, which it does most readily in the sedentary posture, and especially during an expulsive effort. Under these conditions I have known it cause the most violent and painful straining during defæcation, apparently due to the organ being large, tender, and gorged with blood, and partly because it is driven into Douglas's pouch, where it presses on the rectum and interferes with the passage of fæces through it.

From what I have told you you readily gather the circumstances under which the kidney becomes capable of a displacement sufficient to produce pain or discomfort owing to its becoming distended with

Digitized by Google

blood or because its pelvis has been rendered overfull of urine by obstruction of the flow through the vein or ureter. It usually occurs in women, and may appear in an unmarried girl or in a woman after she has borne one or more children or after she has ceased to menstruate, for the reasons I have already given you.

After parturition the tendency of the kidney to drop is much exaggerated by the removal of the support afforded by the pregnant uterus and by the laxity of the abdominal wall. The same condition is favoured by loss of respiratory capacity, flesh and muscular tonicity consequent upon a severe illness or similar period of worry and anxiety to which women are so frequently exposed, or it may result from a change from an active, vigorous life to an indolent physical existence.

We will now consider the *causes of the pain* which sufferers from movable kidney experience. I believe that they are two in number and that the more important one is the engorgement of the organ, which results from its getting into such a relationship with the vena cava that more or less considerable obstruction exists to the escape of venous blood from it. Because of this it becomes swollen, tense, sensitive, and painful. Any jar, as in walking up or down stairs or in taking more violent exercise, exposes it to impact against the ilium or to compression by muscles. Even the alternating compression it is subject to in walking quietly may be so painful that the patient may be unable to flex the hip-joint, but walks with it in a rigid position. The amount of pain and tenderness and the degree of dragging felt in the loin do not, however, bear a necessarily exact direct relationship with the amount of mobility, as a moderate mobility in one patient may be accompanied by very great pain and tenderness, while in another a very free mobility may be present without there being any complaint whatever of pain. The difference in symptoms probably depends on the amount of interference with the passage of blood or urine from the kidney.

I should say that the partial or complete occlusion of the ureter consequent on the misplacement of the organ is comparatively rare, though by no means unfrequently met with. I have an impression that the gastro-intestinal disturbance which is often complained of in cases of movable kidney is due rather to distension of the pelvis of the kidney with urine than to engorgement of its texture with venous blood.

The difficulties that have been experienced in the diagnosis of mobility of the kidney have been due chiefly to the habit of examining patients in the supine posture alone. In this position particularly the kidney causes no pain, and very frequently occupies its best possible position. I know few conditions in which more different opinions are expressed than in this, and often by the most experienced observers. As an instance, I was once asked to remove a calculus from the gall-bladder of a lady by her medical attendant. He told me that his patient had been supposed to suffer from indigestion in the first instance. She was taken up to London to a great authority on the stomach, and was told she had a tumour of the pylorus. Another equally famous man told her that there was no doubt that the lump was a stone in the gall-bladder, and that it was the largest he had ever seen. Both advised operative interference. An examination of the patient showed a freely movable right kidney, which was operated on and secured in position, while to satisfy the puzzled and doubting husband, the abdomen was opened in front, and a normal gall-bladder and stomach were exposed.

I should like to point out to you a condition which I have known to be frequently confused with excessive mobility of the kidney, as well as with stone in this organ, as it is a very common one and as far as I know not generally recognised. It is an obstruction of the upper part of the ascending colon by adhesions; producing distension and hypertrophy of the cæcum. Because of the intimate fixation of the colon to the kidney the patient complains of pains which simulate very closely those of trouble in the kidney. Before I recognised this condition I frequently cut down upon the right kidney expecting to find a stone, and after carefully separating it and its pelvis from surrounding structures and exploring the organ have been much disappointed at finding nothing. To my surprise, however, the patient frequently lost all pain and discomfort. These experiences have occurred to many, and are well recognised. As soon as I became familiar with this state of cæcal obstruction I have called your attention to, I saw that the cases I had operated on as renal were

Digitized by Google

really of this character, and that the extensive separation of the colon from the kidney in the process of exploring that organ thoroughly was sufficient to remove the obstruction. This obstruction is most marked in the upper two inches of the ascending colon, and often involves the hepatic flexure also. The bands of peritoneum which are spoken of as adhesions are attached posteriorly deeply in the loin, and are often very strong.

In a large number of cases they are the relics of an old tubercular peritonitis, as evidenced by the presence of other adhesions and calcareous glands. In many the adhesions seem to have resulted from a peritonitis originating in the appendix. A certain number appear to me to have developed in consequence of a habitual distension of the cæcum as the result of constipation, and probably associated with attacks of inflammation of the wall. In these cases the arrangement is peculiar, since in addition to the bands deep in the loin and continuous with them, a large vascular peritoneal apron has formed having an attachment to the abdominal wall outside the cæcum and colon, and extending inwards over it and being firmly attached to its surface. Its object is clearly to limit the range of movement of the cæcum and to control its distension. These cases may be very chronic, making the patient miserable for a lifetime, or they may appear, apparently for the first time, as acute intestinal obstruction, the cæcum sometimes becoming rapidly gangrenous. I am glad to have an opportunity of calling your attention to these cases as they are very common, and are very easily recognised, while they lend themselves most readily to surgical treatment with the very best of results.

As to the *treatment of movable kidneys*, it must vary with the conditions present in each particular case. As far as I can gather there are two forms of treatment, one adopted, I believe, almost exclusively by the physician, and the other by the surgeon. The former finds a mobile kidney and orders a belt or truss, usually with a large pad in it, while the latter recommends operative interference. I fail to understand how any pad or truss can be of service in retaining the kidney in the loin. I do not mean that a patient may not derive from these things a certain amount of comfort of just such a character as is afforded by the electric belts used by quacks. But beyond that I believe that they exert no useful mechanical function.

When a kidney has once become seriously painful it is unusual to obtain complete relief from any palliative form of treatment, however efficiently carried out, and an operation has to be resorted to sooner or later. In such cases the pressure exerted by any form of pad or truss is out of the question, since the organ is too tender to bear it. I have, however, frequently known them ordered, and at once discarded by the patient. In young subjects an early condition may very often be met successfully by attention to the habits and physique of the patient in the manner I have already indicated, and even later in life a considerable amount of alleviation may be obtained in this way. Clearly the right thing to do is to avoid the occurrence of the mobility altogether by attending to the physique of the child from an early period, and if this be done the individual will certainly escape such a painful and troublesome sequel, as well as from a vast amount of trouble to which these poorly fuelled feeble people are so liable owing to their very unsatisfactory mechanical relationship to their surroundings. This should surely engage the attention of those medical men who are brought into constant relationship with their patients in general practice. Having decided that fixation of the kidney is the only satisfactory means of relieving the patient of pain, the question arises as to how this can be best done. It is now nine years since I published in the 'Transactions of the Clinical Society,' 1892,* a method of fixing the kidney with perfect security in the loin by utilising the capsule. At this time the operation which had recently been adopted was to fix the kidney by buried sutures which passed through its substance, and the results of such treatment were openly acknowledged by many first-rate surgeons to be most unsatisfactory and uncertain. Previously it had been the custom to use temporary sutures. I had myself examined many cases in which this operation had been performed without its affecting the mobility of the organ.

The method I have described, and which I employ, is the following. The kidney is thoroughly exposed through a slightly oblique incision in the loin and freed from any fat that may be present.

* "A Case of Floating Kidney treated by a New Mode of Nephrorraphy."

Digitized by Google

As it projects from the wound the capsule is stripped from its lower two-thirds in the form of six triangles, as shown in Fig. 1, the bases of the triangles being left intact. In order to increase its resisting capacity each is twisted up, when a piece of sterile silk twist is tied securely to it. The kidney is replaced within the abdomen in such a position that little more than half of it is exposed below the last rib. Needles are placed on each position in which it is fixed. The incisions in the muscle wall and skin are then brought accurately together. I have performed this operation a very large number of times, and I have never known the kidney to move from its attachment to the wall. On two occasions I have subsequently had to expose such a kidney once to remove a stone, and the kidney substance and muscle wall were observed to be most intimately united.

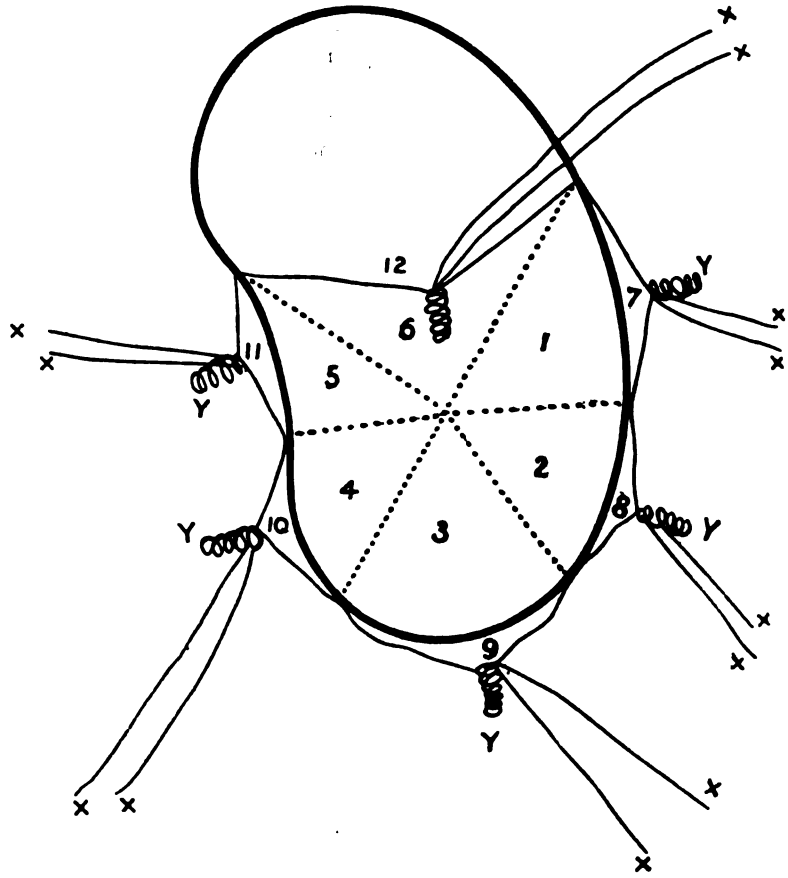


FIG. 1 represents the posterior surface of the kidney, the dotted lines showing the triangular areas (1, 2, 3, 4, 5, and 6) from which the segments of the capsule have been stripped (8, 9, 10, 11, and 12). These are shown with their bases continuous with the kidney and twisted, Y representing the twisted portions, a silk suture, x, being attached to each.

silk thread, and they are passed through the muscles within an inch of their cut margins so as to bring the raw surface of kidney into apposition with the deep surface of the muscle wall freed from fat and fascia. When these sutures have been secured the resistance offered by the capsule is so considerable that it would require the expenditure of a great amount of force to displace the kidney from the

When I have found any considerable hydronephrosis resulting from excessive mobility or from other cause I have employed a method which has been more successful than I ever ventured to expect. It has enabled me to save a number of kidneys which would in the ordinary course of events have been excised. I am strongly of opinion that the operation of excision of the

Digitized by Google

kidney has been undertaken much too frequently and often unnecessarily. I do not think that we should resort to such a serious measure until we have tried every other means possible to save the organ, following the same general principles that guide us in operations on the limbs.

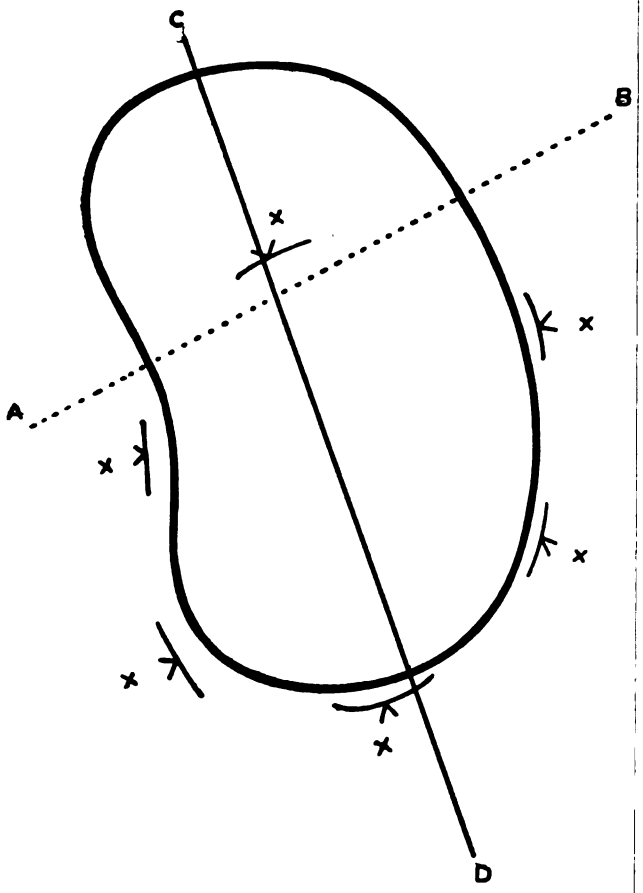


FIG. 2 represents the conditions present after the incision through the muscular wall has been closed. The incision is indicated by the firm line, C D, crossing the kidney from above, downwards and outwards. The dotted line, A B, shows the margin of the thorax.
The several sutures, ×, connecting the capsular segments to the muscle wall are seen emerging from it and tied securely.

The procedure I adopt consists in exposing the hydronephrotic kidney with its pelvis and a considerable length of the ureter by a long oblique lumbar incision, defining its mode of entry into the dilated pelvis. If any vessel is present in this situation over which the ureter is hitched it is ligatured and divided, but such a cause of obstruction is in my experience very rare.

An incision is made along the outer margin of the kidney into the dilated pelvis, and if possible a fine gum-elastic catheter is introduced through it into the ureter, along which it is passed beyond the brim of the true pelvis. In most cases I have found it impossible to introduce the catheter into the ureter in this way, so I have opened the ureter by a vertical incision and passed the catheter through it into the pelvis of the kidney and out through the incision in this organ. The end of the catheter being in the ureter it is forced down beyond the incision in its wall for a considerable distance, when the incision in the ureter is accurately closed by means of a fine silk suture. The incision in the kidney is sutured, a piece of drainage-tube being left in the pelvis, and this with the catheter projects through the skin incision. The catheter is left in the ureter for six weeks or two months, during which time urotropin and sandalwood oil are freely administered. By the use of these drugs the urine remains normal, and the catheter does not become encrusted with any deposits of salts on its surface, but is perfectly sweet and clean when removed at the end of this time.

The drainage-tube is also taken away within a few days and the wound closed.

French and German Methods.—Among the different communications made lately, a certain number show that German and French surgeons are not of the same opinion on certain subjects. Most German surgeons, such as von Bruns, Limon, Czerny and Henle, consider that castration is the best treatment of tuberculosis of the testicle, whereas König, Gussenbauer, and Beir believe in temporization before adopting such vigorous methods. German surgeons prefer vaginal hysterectomy in cancer of the uterus, and most of them believe it is best to defer the operation in appendicitis. Braun and Krause give their statistics in the treatment of epilepsy by extirpation of the cervical sympathetic nerve, and the results adduced seem far from satisfactory. Kocher gives a new series of a thousand operations for goitre with only fourteen deaths.—*Journ. Amer. Med. Assoc.*

Digitized by Google

WITH DR. HALE WHITE IN THE WARDS OF GUY'S HOSPITAL.

(*Concluded from page* 85.)

This man is forty-eight. His persistent albuminuria has led some of you to suggest Bright's disease, but he really has no cardiac, arterial, or retinal signs of this; he has had no œdema, and the pain in the loin points to a calculus in the kidney. It is important to note that his pain is always on the same side. How do you tell whether a stone is in the cortex or the pelvis of the kidney? One point is that the pain is very different. When the stone is in the pelvis of the kidney, the pain shoots down to the inner side of the thigh and down to the testicle or labia, as the case may be. When the stone is in the kidney itself, the pain is a constant boring pain in the renal region.

The very interesting case has been recently recorded by Mr. Mansell Moullin of a patient who had pain, and was operated on for appendix trouble, but got no better. Then he was radiographed, a stone in the cortex of the right kidney was found and removed. After this he had no pain.

One interesting point in connection with this subject is how, after any part of the body has been scarred by some internal disease, it will ache long after the cause has been removed. A fellow student of mine had all the typical symptoms of renal calculus—hæmaturia, and so on,—and after two years he passed a calculus, and he still had pain in the lumbar region, but the character of the pain altered. When the calculus was present exercise caused the pain, and long walks were impossible; but after his calculus was passed, the pain was only present when he was tired and weary, and even if then he lay down because he was tired, the pain was very bad so long as he was weary, but if he went to Switzerland and got into good health, he could climb mountains without feeling pain. Previously to the stone being passed he did not feel the pain particularly badly when he was tired, but if he took violent exertion he had pain. That sort of thing lasted three or four years and then slowly disappeared. The same may be true of pleural adhesions; they cause an aching when the owner of them is tired, otherwise they give no trouble. The mystery is the large number of people who have every symptom of calculus, and yet none is found; they may even have hæmaturia. But we have often discussed these cases.

Here is a case which will repay you for going over. It is that of a man æt. 45. The points about him are: that seven years ago he felt a stiffness in the left leg; then the left arm became weaker, and he felt numbness in his fingers, with the exception of the thumb and index finger. Then he says the right leg and arm became affected, but to a much less extent. Four weeks ago he had pain over the cæcum. He was told he had rheumatic gout, and he went to the out-patient department and was admitted. Three weeks ago he noticed that when once he had the desire to micturate he had to pass his water immediately, otherwise he could not hold out. Sometimes he had to get up and pass it two or three times in the night; but he never passed it in bed. Now we will see him walk. You will notice he holds the left leg rigid. He has ankle-clonus in both legs, and exaggerated knee-jerks. An extensor planter reflex is obtained on either side. Therefore the points to remember about him are: that he is stiff in both legs, his knee-jerk is exaggerated on both sides, there is clonus in both ankles, and he has got an extensor instead of a flexor response of the great toe. A combination of symptoms like that shows that he has got lateral sclerosis, and the next thing to determine is how far up it extends. When I examined the arms I could not make out much the matter with them; but he complains of slight loss of power and a little numbness in the left hand, so slight that we hardly know what weight to give to it. I did not think that his elbow jerks were increased. The wrist-jerks are present. That, with the difficulty in retaining his water, is the whole case. The point to seize on is that he has some lateral sclerosis, and that it has not extended so high up in the cord as to affect the pyramidal tracts where they are distributed to the arm. Now, if he has got lateral sclerosis, what is it due to? It means that below the lesion the axons which form the pyramidal tracts are cut off from the cells in the cortex, and clinically a very common cause of this sort of condition, when both lateral columns are affected, is a syphylitic myelitis. For some reason which we do not understand, syphilitic myelitis is very much more common in the lumbar region than in any other. For some reason, of which we are equally ignorant, it fre-

Digitized by Google

quently spares the sensory tracts and commonly affects the motor tracts. So if we imagine that he has got an incomplete syphilitic myelitis somewhere about the lower part of the cord, and that below that part, because of it, secondary degeneration has taken place in the two pyramidal tracts, we shall explain the condition of the legs. That, I think, is the most likely view. That will explain why it is that he cannot hold his water properly. Impairment of micturation is so slight that the bladder centre itself is all right; but this man wants to empty his bladder too often because he cannot send down controlling impulses to prevent him doing so. Therefore when the bladder gets to the point when it reflexly wants to be emptied, the urine is likely to be reflexly shot out, because, owing to the disease in the man's cord, he cannot get impulses down to restrain it. The reason I say syphilitic is, that these slight cases of myelitis, sparing the sensory and affecting the motor tracts, producing symptoms of lateral sclerosis, and very little impairment of bladder, are nearly always syphilitic. The right treatment is to give him iodide of potassium and mercury. You will say how will that do any good if the cord is destroyed? I do not think it can make him a new cord, but if we can at once begin to promptly treat his syphilis, we may prevent his getting any more myelitis, and we may also put him into the best condition for getting his inflammatory products of the present myelitis absorbed. This state of things is very common, and we have had two or three patients with it in at once. I do not want you to think it is a gummatous condition, but it is that people who have had syphilis are particularly liable to this form of myelitis, and you cannot cure it absolutely by giving iodide. But you may prevent the mischief spreading. Now you may say quite rightly that I have not explained the condition in the hands. He complains of numbness there. He says this hand got weak. A strong reason why I treat it on the idea that it is syphilitic myelitis is that it is the only thing which we can do anything for, and we should leave the arms out of the diagnosis as far as the treatment is concerned. If the condition of the arms is correct, and the numbness gets worse, and he loses more power, then it becomes extremely likely that he has general paralysis of the insane, which again, like the myelitis, is a secondary manifestation of syphilis. It is not gummatous, but a person who has had syphilis is more likely to get general paralysis than other people. It reminds me of an axiom, that if you have any obscure nervous disease which is difficult to fit into any known disease—it is difficult here to fit in the two arms being affected with the legs,—it is very likely due to general paralysis of the insane, and particularly so if the insensity is increasing more quickly than you would expect in any other condition. Very rapid symptoms, simulating what would be a chronic disease, should always suggest to you that it may be general paralysis. If a man comes to you and you are tempted to say "what an extraordinary thing he has got locomotor ataxy so quickly," you may feel fairly confident that locomotor ataxy is the beginning of general paralysis.

Here is a man æt. 32. Two years ago he began to have frequent coughs, lost weight, and became weak. Then he got somewhat better, coughed up blood-stained sputum, and then brought up a quart of blood and sputum. He says that now he brings up a pint of sputum a day. You see him now with a flushed face, and his fingers are clubbed. Notice the way in which the finger-nail is bent over the end of the finger, and how much substance of the finger is immediately underneath the nail. What are the causes of clubbing of the fingers? They are pulmonary disease and congenital heart disease. It is very important to remember that. The two forms of pulmonary disease which commonly cause it are phthisis and empyema. A number of physical signs have been made out in him. The most striking point is that the air does not enter the left side as well as it does the other. You can also see that the movements of the two sides of the chest during inspiration are not equal. Moreover the left side of the chest appears flattened. Do you think the impaired entry of air, and the impaired visible movement, and the flattening necessarily mean fibroid lung and nothing else? Of course a thickened pleura is possible. Now these signs of dulness, impaired entry of air, impaired movement, falling in of the chest, may be due to fibroid lung, but they may be equally due to thickened pleura, because the pleura then contracts down on to the lungs. The systolic retraction at the apex shows that he has got adhesions. You will notice that the apex of the heart is

Digitized by Google

displaced outwards to the left; that is probably because the adhesions are dragging it out of place. I think he has got a fibroid lung as well, but I want you to think of the possibility of these things being due to thickened pleura also. The point of interest I want to bring before you is this, that after all he has not so very much wasted, as phthisical patients go; although he has been ill for two years, and has brought up quantities of sputum and blood, yet he has gone on with his work, and is no more wasted than you see. People with a large extent of physical signs in phthisis often do extremely well, because a large amount of physical signs may mean that the condition is tending to heal itself. A quantity of new fibroid tissue gives abundant physical signs, but on the other hand shuts in the bacilli. People with much fibrosis, like this man, are therefore those who improve. He has no high temperature. Would you expect a high temperature from the physical signs? No, because we think the pleura is fibroid, and there is fibrosis in the lung, and it shuts in the bacilli and prevents absorption of their toxic products. There is every indication here that the bacilli are getting shut up, and that is exactly what we want, so that their toxins cannot be absorbed. The worst case I know, as far as physical signs go, is that of a man who has great falling in of the side, and his whole chest is perfectly dull. There is evidence in this patient that the whole lung is little more than fibrosed tissue, and so no absorption of toxic products can take place. But he can walk five or six miles, and although he cannot work much he enjoys life. Such patients do not waste much, they have not much temperature, they have a fairly uniform pulse, and they do well. This man's sputum has not yet been examined for bacilli; probably they are very few. With regard to prognosis, not only have you to determine how far the lung is fibrous, but another great point is to ascertain how far the patient takes his food and digests it, because we believe, and quite rightly, that the best cure for phthisis is to get the general health of the body into the highest possible state, and that, of course, cannot be done unless plenty of food is taken, and is properly digested. We learn from him that he is not sick, has no diarrhœa, and that he has a good appetite. This is then the most hopeful case of phthisis we have had in the hospital for a long time, and it is the best to illustrate the points which you have to look for in forming a prognosis. You need to know whether the man is much poisoned by the toxins, and whether his gastro-intestinal tract is working properly. Few cases of phthisis die from the local effects of the disease in the lung, they die of toxæmia or starvation. We know this man has not got much toxæmia, because his temperature is not up, his pulse is good, he does not sweat, is not anæmic, and he has not lost flesh, and on examining him we find the reasons for his not having much toxæmia, for the physical signs show there is much fibrosis, and this is probably shutting in the bacilli. It has been shown that he takes his food well, and absorbs it, and so his resisting power is increased. After he has rested a little longer it will be well to get him into the country. I may say that the profuse expectoration which you notice he has tends to show that his tubes are dilated, probably secondarily to the pulmonary fibrosis. It is a point to remember, that when the tubes are dilated as a result of phthisis it is very rare for the sputum to be foul.

The next patient is a boy of 16, with mitral stenosis and aortic regurgitation. He has been having daily a bath of a temperature of between 95 and 97, and lasting a quarter of an hour, and says he feels very much better for it. Of course we all know that a bath braces us up. I suggested that he should have a bath every day, and he has been given one, with a great deal of apparent improvement of his feeling of well-being. These cardiac cases get an oppressive pain in the chest, and that, in this patient, seems to have been relieved by the bath. This boy was at first reluctant to have the bath, so that in this instance it is hardly a case of the "wish being father to the thought." Leonard Hill found that when a chloralised rabbit was held in the vertical position its blood-pressure steadily fell, but if the body was plunged into water the pressure at once regained normal, or nearly normal. The probable explanation of that is, that the benefit is due to the increased pressure in the abdomen being exerted on the abdominal vessels. There may be something of the same sort in these patients with heart disease who feels better for baths. I was induced to try baths, because patients with heart disease who go to Nauheim have baths, and often seem to be benefited by them.

Digitized by Google

This boy, æt. 12, came in with general anasarca, due to acute nephritis. Acute nephritis is a rare disease, and tends to relapse. This boy was in the hospital for nine weeks nine years ago, and at that time he had acute nephritis. When he was sent out he was apparently perfectly well. His nephritis then was not scarlatinal, and there is no evidence that it is scarlatinal now, or he would not be in hospital. Nine weeks ago he began to swell about the trunk, face, and neck. As he got no better he came to the out-patient department, and has been attending ever since. He noticed that his urine was very thick, and the chief interest of the case lies in the urine. There is a great deal of albumin in it, and it is of 1040 specific gravity. Probably also there will be found to be many epithelial casts. There is a high percentage of urea present, but it will very likely be found that he is not passing much urine. You will remember I said he had an attack nine years ago, and now he comes with the same condition, showing how liable it is to recur. Even on looking at the boy we could almost decide that he must have Bright's disease, for such a large amount of pale œdema could hardly be anything else; there is too much facial œdema for heart disease or bronchitis. Notice particularly the extreme degree of facial œdema, and there is also some sub-conjunctival œdema. The œdema is also very evident in his legs, scrotum, and feet. The pulse is not of high tension. There is no pain in the loins, and there is no rise of temperature. What do you think his kidneys would look like? They would be found to be very large, and the change would be tubal, and they would now probably be pale in colour. If seen six weeks ago they would have been engorged with blood from the acute attack, and now that stage would be subsiding. All the tubes are completely blocked up with fatty degenerated epithelial cells. You would also expect to get tube-casts in the urine for the same reason. The boy is probably passing very little urine, and consequently what he does pass is of high specific gravity. What should we do for him? We should give him purgatives and diaphoretics. I think the bowels should be kept open, because you can get much fluid to pass by means of powerful purgatives. We therefore select a purgative which will produce a watery motion, such for instance as jalap or scammony or magnesium sulphate. We shall also make him perspire, and for this purpose a hot-air bath is the best. The œdema which you see outside his body is only an expression of what is probably also inside. In his lungs there is fluid at both bases, therefore the more we can make him sweat the better. He should have a hot bath at least once a day. Do not give him pilocarpin unless absolutely necessary, for it causes much secretion of fluid into the bronchial tubes, and this, added to the already present pulmonary œdema, may make his breathing dangerously difficult.

The girl in this bed, whom we will not now examine, so as not to disturb her, shows how chorea is wrapped up with rheumatism, and how common rheumatic sore throats are. She has had "growing pains"—which mean rheumatism,—and comes in with chorea. We can detect a murmur, and you will be prepared to hear that it is a mitral murmur, because it nearly always is in chorea. More frequently it is systolic than diastolic, and it is systolic here; a soft, not a rasping, systolic murmur, audible at the apex, and therefore due to mitral regurgitation. Almost invariably it is not associated with any signs of backward pressure. So when you make a post-mortem on these cases you find the mitral valve is all but competent, and only just a little fringe of vegetations on the upper surface of it. It is very important you should remember this, because if you do not, you get to think of the heart disease which goes with chorea as such a serious matter, which it is not. Indeed, it seems almost as if when a person gets chorea it protects her at that time from the worst form of rheumatic heart disease, because almost invariably the murmur found with chorea is soft apical systolic murmur, without any other signs of heart disease. If it is not that, it is an equally inoffensive presystolic murmur. It is highly exceptional to see cases of chorea with such bad heart disease that they are waterlogged, and it is also exceptional to see cases of chorea with pericarditis. That girl has the usual cardiac condition which is associated with chorea. She will probably get another attack of chorea. At what age can we tell her mother she will be free from it? I think we may safely say from twenty-three to twenty-four years of age is the limit. The prognostic feature of even very bad attacks of chorea is, that you hold out two hopes. In the first place, it virtually never kills of itself; and in

Digitized by Google

the second place the sufferers from it always outgrow it, and it hardly ever exists after twenty-three years of age; often such patients do not have any more after eighteen. Sometimes, however, it does kill, and then the death occurs from sheer exhaustion, the patients knocking themselves about; they will sometimes throw themselves on to the floor and injure themselves. They die quite rarely from hyperpyrexia. A bad case of chorea looks most alarming, but you are able to tell the friends that she will grow out of it.

Is there any other cause for it besides rheumatic fever? It may occur in pregnancy, but it may then, for all we know, be another disease, because in many women who have it in pregnancy there has been no rheumatism. It generally disappears at about the seventh month of pregnancy. An important question in this connection is, Would you induce premature labour for chorea? I answer, it is probably never required. I had a very bad case of chorea in pregnancy in Miriam Ward. We put that particular patient I speak of in No. 17 bed, as it is in a corner; screened her off, and gave her bromide and chloral, and kept her quiet, and at the seventh month all the chorea disappeared, and she was confined normally at the ninth month. It looked very alarming to see her throwing herself violently about the bed month after month, and many thought that surely premature labour must become necessary, and that the confident hopeful prognosis must turn out to be wrong.

A striking thing about chorea, apart from the violent movements, is the paresis. There are two factors at the bottom of chorea; one is the irregularity and violence of the movements, which, of course, is obvious, and the other, which not being obvious is not often noticed, is paresis. Occasionally the paresis is marked, and the movements slight, but in the ordinary case the movements are violent and the paresis slight. Now, paralytic chorea is an affection about which it is very easy to make mistakes in diagnosis. If you get a child—generally a female child—with a rheumatic history, who has loss of power, the cause of which you cannot quite make out, it is a very good rule to wonder whether it is not a case of paralytic chorea. And if there is also a murmur present, I should say you should certainly think of that possibility. You will remember a case in point in No. 16 Bed not long ago. Yet these cases also get well.

What is the best treatment in chorea? Rest, and leaving the patient alone quietly. In the matter of drugs, arsenic is the fashion at present, but my own impression is that drugs do very little. Since I have been at Guy's there have been three periods of about equal length in this matter. For many years all the cases of chorea were given zinc. Zinc, however, is never prescribed now. Then for a long time all these cases had iron; and now for a long time they have had arsenic. I have not been able to detect any difference in the result. Some years ago a committee collected thousands of cases grouped according to the drugs administered, and it was shown that there was little to be said in favour of one drug as compared with another. So I have a strong suspicion that it is the rest which does the good. As a rule, arsenic improves the health a little; and if people want to give arsenic I never think it is worth while to withhold it, but I am very sceptical whether much good can be traced to its use. You must feed the patients well, and often massage is of service.

Idiosyncrasy to Quinine.—That this hereditary predisposition to the effects of certain drugs has been recognised before is well known by theurapeutists. As long ago as 1879 King reported an observation of this character in 'Vierteljahrschrift für Dermatologie und Syphilis.' Sometimes the skin lesions which are produced by quinine are vesicular, pustular, or papular; and Grissac, in 1876, in his brochure upon 'Quinine Eruptions," states that they may be purpuric, roseolar, scarlatiniform, or eczematous. Thus Galloway reported scarlatiniform erythema as long ago as 1869. The development of this rash, particularly in the case of a child, might very readily lead to a mistake in diagnosis of scarlet fever in much the same manner as the morbilliform rash sometimes produced by antipyrin and other coal-tar products misleads the physician into a diagnosis of measles.—H. A. HARE, M.D., *Therapeutic Gazette.*

Digitized by Google

THE OPHTHALMOSCOPIC CHANGES MET WITH IN BRIGHT'S DISEASE.

A clinical lecture delivered at University College Hospital, March, 1901.

By PERCY FLEMMING, F.R C.S.,

Assistant Ophthalmic Surgeon to the Hospital and to the Royal London Ophthalmic Hospital.

I WANT to-day to speak about the ophthalmoscopic changes in Bright's disease, partly because there are several cases in the wards just now, and partly because such cases afford one of the best examples of the value of the use of the ophthalmoscope in general medicine. The ophthalmoscope is a help in investigating a group of cases admittedly difficult of diagnosis, and it is also an important aid in estimating the prognosis. Bright's disease, of course, is not merely a disease of the kidneys, but one which has far-reaching effects, and these effects are felt most by the vascular and nervous tissues. It is on account of this that the ophthalmoscope is useful in investigating these cases, the retinal vessels and the optic nerve being the only vessels and nerves which can be seen during life. We will first of all take the changes to be met with in the retinal vessels in Bright's disease. The simplest way is to consider first the post-mortem changes found in the retinal vessels, and in the second place to see whether those changes can be made out with the ophthalmoscope. The changes that occur in the vessels are of two kinds. One is a general change, the retinal vessels being much thickened. The thickening of the walls is either of a hyaline character or due to cell infiltration. The other change is a local one, affecting the vessels here and there, and is of the nature of endarteritis, the lumen of the vessels being narrowed. This section, which I will pass round, shows the hyaline thickening in a very marked degree. Can these changes, found post mortem, be made out with the ophthalmoscope during life? In the first place the vessels are frequently found to be narrowed, or rather one should say that the blood-stream appears to be small, because, as a matter of fact, in health one never actually sees the retinal vessels, the walls of these being transparent. In the second place the hyaline thickening of the arteries appears with the ophthalmoscope as an accentuation of the light reflex or light streak which is normally present on the arteries, and in more advanced cases the degenerated vessel may have a bright "silver wire" appearance. There is a good example of this shiny appearance of the artery in the left eye to be seen in Ward 3, the patient being a woman with puerperal eclampsia. Further, the effect produced by such a thickened degenerated artery on a neighbouring vein, is characteristic. Where one of these thickened arteries crosses superficially to a vein, the thickened artery will affect the thin-walled underlying vein. The earliest change is a slight displacement of the vein to one side, which, little by little, increases, so that in a markedly degenerated artery the latter will press on the vein and interfere with the blood-current, the vein on the peripheral side to the pressure being distinctly distended. Another change that has to be noted is that the walls of the vessels may actually become visible owing to the thickening, so that the columns of blood are seen to be bordered by white lines of varying thickness; this is also very well shown in the case to which I have referred you. Later, too, the vessels, especially the arteries, will become tortuous. I once observed very tortuous arteries in a case of Bright's disease, after the subsidence of very marked general œdema; the tortuosity being perhaps due to the previously swollen and distended retina having stretched the vessels. But too much stress must not be laid on mere tortuosity of the vessels alone. It is doubtful at what stage in the course of the kidney disease such changes as these appear, but they are important indications of general arterial disease, and as such afford an important prognostic indication, for patients that have these diseased vessels are very liable subsequently to have apoplectic attacks. Marcus Gunn, in a paper published eighteen months ago, gave notes of eighteen cases that showed these changes and their subsequent history; it was found that a large proportion died of apoplexy subsequently. Of three cases that I have observed, death from apoplexy occurred seven, nine, and thirteen months respectively after the changes had been noted.

Another retinal change to note is the occurrence of hæmorrhages. These hæmorrhages vary in shape and size according to the layer of the retina in which

Digitized by Google

they occur; if they occur in the nerve-fibre layer, they will be long, streaky, flame-shaped, as the effused blood will run in between the nerve-fibres separating them; if the hæmorrhage occur in the deeper parts of the retina, they will appear as minute spots or rounded areas of greater or less size. As a rare occurrence the effused blood may rupture the membrana interna of the retina, and bursting through into the vitreous, cause extensive vitreous hæmorrhage with great loss of sight; and as a still rarer occurrence, such hæmorrhage into the vitreous may become the seat of connective tissue formation. There is a specimen showing this condition in the Museum.

So far I have said nothing about "albuminuric retinitis" proper, because I was anxious to review in the first place those changes that are not so well known. Albuminuric retinitis is used in a general way as a descriptive term for various conditions found in the retina in Bright's disease. These conditions result from several causes, such as inflammation, hæmorrhage, degeneration, etc., so that any one case of albuminuric retinitis will be made up of several factors, any one of which may be more marked than the others; and it is best in describing a case to describe the appearances in detail, without designating the case by a particular name, such as degenerative, exudative, inflammatory.

Taking the retinitis first, the earliest sign of this is œdema of the retina. The presence of œdema has the effect of modifying the ordinary bright fundus reflex, so that in places, especially at the posterior pole, a greyish appearance is observed. Later white spots appear, and these white spots are most characteristic of albuminuric retinitis. These spots vary both in their number and size and situation, and are due to different anatomical conditions. Some spots have soft edges, and have a woolly appearance, and are situated in the deeper layers of the retina, so that blood-vessels may be observed to pass over them. These spots are due to a fibrous exudation into the internuclear layer, which exudation may subsequently undergo fatty degeneration, and may become absorbed, so that such spots as these are not necessarily permanent features in the case. This section shows well such a fibrinous exudation. Other spots are due to degeneration of the nerve-fibres and the supporting Müller's fibres of the retina, and have a much harder, whiter appearance than the former spots, and, being the result of degeneration, are more permanent and lasting than the other kind. Such thickening of Muller's fibres is seen in this section. Typically these spots form a star-shaped or spoke-shaped figure at the macula, and this appearance is one of the most prominent features in the picture of albuminuric retinitis. Other causes of white spots are hæmorrhages which have undergone more or less complete absorption. Later on in the case pigmented spots may appear, and these pigmented spots or patches in Bright's disease in some cases are due to a proliferation of the pigment epithelium situated between the bacillary layer and choroid. In other cases definite choroiditis occurs. The second factor in albuminuric retinitis is optic neuritis or papillitis, and in any one case this may be the most marked feature in the ophthalmoscopic picture. But usually the papillitis is of slight amount. In such cases it is commonly associated with retinitis, but there will be a more or less clear area of retina between the swelling of the papilla and the central star-shaped figure at the macula, a point that is of importance in distinguishing this condition from neuro-retinitis due to organic brain disease, in which case, as a rule, the swelling of the papilla passes over and becomes continuous with the retinal exudation at the macula. The third factor in albuminuric retinitis is hæmorrhage, which may have the same characters as those mentioned in the earlier part of the lecture. These three factors —retinitis, papillitis, and hæmorrhage, occurring in various combinations, make up the condition recognised as albuminuric retinitis. And in any one case the retinitis may be the prominent feature, in another the papillitis, and in another the hæmorrhage; and on such grounds varieties of albuminuric retinitis are described such as the neuritic, hæmorrhagic, and inflammatory. It is an interesting question, but impossible to answer at present, as to how far these retinal changes are the direct sequence and the result of the vascular changes described in the earlier part of the lecture. For instance, it is possible to regard the interference of the blood-supply, the result of the arterial disease, with its resulting pressure on the vein, as causing degenerative changes in the retina, such changes constituting albuminuric retinitis.

Digitized by Google

Looking now at the matter clinically, the first point to inquire into is, how frequently are these retinal changes met with in Bright's disease? Numbers given by authorities vary very much—from 6 to 30 per cent.—if one includes the vascular changes, I am sure that the number should be much nearer 30 than 6 per cent. Then as to the particular kind of Bright's disease in which these changes are met with; they may be met with in any kind of Bright's disease, with the one exception of the kidney disease consecutive to bladder trouble, but they are much the most commonly met with in late granular contracted kidney. In parenchymatous nephritis the changes are found, especially in connection with pregnancy. They have been found also in children, but this is rare, and then usually as a sequel of scarlet fever. I think it impossible to say that any one form of Bright's disease is associated with one particular variety of albuminuric retinitis, although it has been recently stated that the degenerative variety is particularly associated with the granular contracted kidney, and the inflammatory exudative types with parenchymatous nephritis.

As regards prognosis, the importance of noting all these changes arises from the fact that they usually occur late on in the course of granular contracted kidney, and the general prognosis is therefore bad, so that a patient with granular contracted kidney and double albuminuric retinitis will in all probability die in the course of twelve months, and very likely much sooner. On the other hand, the occurrence of albuminuric retinitis in cases in which the primary disease is amenable to treatment—such, for example, as the Bright's disease associated with pregnancy—does not influence the prognosis at all. Recent statistics in connection with the prognosis of albuminuric retinitis have shown very clearly how much more favourable it is in cases occurring in private practice, where the patients can be looked after better than in hospital practice, where the prognosis in Bright's disease is notoriously very bad. These two ophthalmoscopic drawings represent the improvement that may take place when the cause is removable, as in pregnancy. You will see how many of the white patches of hæmorrhage have disappeared, and along with this objective improvement the vision improved from $\frac{6}{60}$ J. 12 to $\frac{6}{9}$ J1. As regards the treatment there is little to be said; the treatment being mainly that of the general disease, but the eyes should be, of course, rested, and shaded from the light by dark glasses. In one set of circumstances, viz. the albuminuric retinitis of pregnancy, it is an important point in the treatment to determine whether the pregnancy should be terminated or not, because the changes in the retina may quickly subside if the pregnancy be brought to an end. It is quite clear, therefore, that the occurrence of albuminuric retinitis with albuminuria in pregnancy is an important factor in recommending the induction of premature labour.

Operation for Vesico-vaginal Fistulæ.—Grassokovkozky publishes an ingenious operation for the cure of vesico-vaginal fistula which was successfully tried in five cases. The usual methods of producing a new raw edge of the fistula are objectionable, because considerable tissue must be removed, and the difficulty, if not impossibility, of accurately splitting the vesico-vaginal septum. He proceeds as follows: A curved needle, armed with a strong silk ligature, enters the edge of the fistula at the junction of vesical and vaginal mucous membranes, and emerges in bladder. Of these ligatures about six are required, the number varying with the size of the fistula. When in position, traction is made upon these ligatures toward the operator, and with a small scalpel the septum is split the whole circumference of the fistula. The traction upon the ligatures produces a funnel-shaped opening, and, according to the author, the splitting of the septum, usually a difficult and tedious step, is surprisingly simple and easy. This being completed, the third step of the operation consists in the introduction of an eyelet probe (forceps) into the urethra, entering the bladder and emerging from the fistula into the vagina. To this probe the six ligatures are fastened, whereupon it is withdrawn and the ligatures now project from the urethra. As the ligatures only comprise the vesical membrane, traction upon them must necessarily constrict the vesical lumen of the fistula and also project the membrane into the bladder. The final and remaining step of the operation consists in the closure of the vaginal opening with a deep interrupted suture, and the withdrawal of the six temporary sutures from the urethra.—*American Journ. of Obstet.*

Digitized by Google

A CASE OF ACUTE GLANDERS.

By WALTER C. C. PAKES, D.P.H.,
Bacteriologist to Guy's Hospital.

MOST cases of glanders in the human being are associated with the same disease in the horse, and it is generally possible to trace the disease to some equine source. The importance of the following case lies in the fact that the patient was a solicitor's clerk, and so far as the history can be traced he never had anything whatever to do with horses. This latter statement appears to be quite correct, since the medical practitioner in attendance was able to obtain a very full history from his relatives.

Percy B—, æt. 28, fell ill on March 14th with what was supposed to be influenzal catarrh, after having had a long journey by rail on a cold day. When he first complained of feeling ill the chief symptom was neuralgia of the left eyebrow; this, together with the upper left eyelid, was distinctly swollen. His temperature was on this day 102°, and it continued to rise till the 17th, when it reached 104°. During these three days the inflammation had extended to the eye itself. On the 19th the right eyebrow and forehead had the appearance of erysipelas, but several bullæ were observed over the area of affected skin. On the 20th both eyes were equally affected, and he began to cough, but examination showed no physical signs. On the 21st pustules began to appear on the left side of the forehead. By this time he was delirious, and cried out with pain when his joints or limbs were touched.

On this day (the 21st) the examination of the skin showed the following conditions:—There was an extensive pustular eruption on the body, arms, legs, and chin. The rash on the body was mainly confined to the chest, there being only a few spots on the abdomen. It was present on the arms, more especially on the extensor surfaces, and there were a few spots on the backs of the hands. There were also some pustules on the upper and outer parts of the thighs. There was neither rash nor pustule on the scalp, in the mouth, or on the palate.

The rash had the following character:—Small pustules varying in size from a quarter-inch diameter to minute spots were present. They were tense and filled with pus. Some had broken, and left a depressed red mark, with sharply sloping sides. Some of the pustules were distinctly umbilicated. The epithelium over the pustule was very friable, and ruptured with but little friction.

Right eye.—A large surface above the right eye was in "a carbuncular condition." It was covered with dead skin, which could be easily rubbed off. The slough was soft, but no bare bone could be felt. At the inner angle of the eye there was an older slough, from under which pus could be squeezed. The probe introduced here passed upward for about three inches. The eye was not disorganised, though there was considerable chemosis.

Left eye.—There was marked chemosis and a large abscess occupying the supra-ciliary region. The pus from the abscess was thick and curdy, but no bare bone could be felt.

On the 25th he died, and a post-mortem examination revealed nothing which could throw any light upon the case.

During life the pus obtained from the sinus at the inner angle of the right eye was examined bacteriologically, and was found to contain a pure culture of the *B. mallei*. This organism gave all the typical reactions, and after injection into the subcutaneous tissues of a male guinea-pig produced the typical testicular lesions, and killed the guinea-pig in seven days.

It is true that glanders was suspected during life, chiefly on account of the pustular rash, but this diagnosis was very uncertain, and it was only by the bacteriological examination that the true nature of the case was arrived at.

As has been previously mentioned, the patient had no connection with horses, and the source of the infection will remain a mystery, but the case points to the possibility of glanders being contracted by the "man in the street." It is not impossible also that some of the cases of fatal pustular conditions may be shown by bacteriological examination to be glanders.

In conclusion, I must express my thanks to Dr. F. Taylor for his kindness in allowing me to publish this case.

A WARM bath just before going to bed tends to allay the nervous irritability which prevents sleep in children, whether caused by temper or fatigue.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 450. WEDNESDAY, JUNE 12. 1901. Vol. XVIII. No. 8.

CONTENTS.

PAGE

***Some Remarks on the Causation and Diagnosis of Acute Intestinal Obstruction.** By J. H. BRYANT, M.D., F.R.C.P. 113

***Four Clinical Lectures on Some Affections of the Kidney.** By A. H. TUBBY, M.S., F.R.C.S. Lecture II 122

***With Mr. Jonathan Hutchinson at the Medical Graduates' College and Polyclinic** 126

Errors in Laboratory Diagnosis ... 122

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

SOME REMARKS* ON THE CAUSATION AND DIAGNOSIS OF ACUTE INTESTINAL OBSTRUCTION.

By J. H. BRYANT, M.D., F.R.C.P.

MR. PRESIDENT AND GENTLEMEN,—I feel it a very great honour to be here this evening to address this Society, and thank you for the compliment you have paid me. When your Hon. Secretary, Dr. Dees, asked me to read a paper, I was at first at a loss to know what subject to choose; I thought, however, that some remarks on the causation and diagnosis of acute intestinal obstruction would serve the purpose, and would be of general interest. I was also influenced in my choice by the recollection of several extraordinary cases which have come under my notice, and by the collection of splendid specimens we have in the museum at Guy's Hospital, many of which, and photographs of others, I am showing here this evening to illustrate my remarks. I do not claim originality in this paper, as the subject has been so thoroughly worked out, first of all by Dr. Hilton Fagge, and more recently by Sir Frederick Treves, but I hope to make it practical and possibly interesting by frequent allusions to actual cases.

Acute intestinal obstruction is a condition which may be brought about by a great variety of causes. It may be produced by the presence of foreign bodies blocking up the lumen of some part of the gut, it may be caused by pathological conditions of the wall of the gut itself, *e. g.* the various forms of stricture of the bowel, intussusception, etc., and it may be the result of external conditions involving the gut, *e. g.* bands, etc.

I propose, therefore, to classify the causes of acute intestinal obstruction under these headings, *e. g.*:

1. *Conditions inside the gut.*—Foreign bodies.
2. *Pathological conditions involving the gut itself.*—

* Delivered before the Wimbledon Medical Society.

Digitized by Google

(*a*) Strictures becoming acutely obstructed; (*b*) intussusception; (*c*) volvulus.

3. *Pathological conditions external to but involving the gut.*—(*a*) Strangulation by bands; (*b*) strangulation through orifices and internal herniæ; (*c*) kinking of the gut; (*d*) tumours pressing on the gut.

I will now take each heading separately in the order in which I have given them to you, so that first of all I will consider *foreign bodies* as a cause of acute intestinal obstruction. Foreign bodies may be defined as substances which are not acted on by the digestive fluids of the alimentary tract. They may be composed of mineral or vegetable substances, *e. g.*:—(1) Gall-stones. (2) Masses of phosphates, *e. g.* of calcium, magnesium, etc. (3) Magnesium salts which have been taken in excess as medicines. (4) Masses of vegetable fibre, *e. g.* the indigestible part of oats, cinnamon bark, cocoa-nut fibre, etc.

Of these, gall-stones are by far the commonest as a cause. They may block any part of the small intestine, from the upper part of the jejunum to the lower part of the ileum, but most frequently the ileo-cæcal region is the portion involved. They must, therefore, be of considerable size, and they never get into the intestine through the bile-duct; they are much too large to take that course; they always ulcerate through from the gall-bladder into the duodenum, and may leave a cholecysto-duodenal fistula. Before this takes place, there is usually some local peritonitis, which binds the gall-bladder to the duodenum, so that a general peritonitis is in this way avoided when the walls of the gall-bladder and duodenum are ulcerated through. Peritonitis plays a most important part in acute intestinal obstruction, especially in the production of it, as will be well seen when strangulation by bands, kinking, etc., are considered.

I have here a specimen and a photograph of it (Fig. 1) showing a cholecysto-duodenal fistula and a gallstone impacted in the ileum. The fistula is large enough to admit the middle finger.

The patient was a woman æt. 59, who was admitted under the care of Dr. Hale White for intestinal obstruction. For six days she had suffered from abdominal pains, vomiting, and constipation. She was operated on two days after admission, laparotomy being performed, and an artificial anus made in the small intestine. She died six hours afterwards, and the gall-stone was found impacted i the ileum thirty-three inches above the ileo-cæcal valve.

Here is a gall-stone which ulcerated through the gall-bladder into the duodenum, and caused acute intestinal obstruction and death. It measured one and three quarter inches in length, and a little more than one inch in its transverse diameter.

The patient was a very stout woman æt. 59, who three months before her fatal illness suffered from pyrexia and pain in the side. Vomiting came on six days before death, but there was neither abdominal pain nor distension. At the necropsy the gall-stone was found about thirty inches below the pylorus firmly impacted in the jejunum. The duodenum and gall-bladder were firmly adherent to each other, and there was a communication between these two organs just below the biliary papilla.

These two cases illustrate the point I drew attention to, viz. that either the upper or the lower part of the small intestine may be obstructed, for in the first-mentioned case the calculus obstructed the ileum thirty-three inches above the ileo-cæcal valve, and in the second case the obstruction was only thirty-three inches below the pylorus

Obstruction by gall-stones occurs much more frequently in women than in men, and in nearly 85 per cent. of the cases the age of the patient is over fifty. In some of the cases a previous history of gall-stones, jaundice, or biliary colic may be obtained. The patients are as a rule stout. Vomiting and abdominal pain occur very early after the onset, and are usually severe. Fæcal vomiting is frequent and occurs early. Jaundice is rarely present, and it is most unusual to be able to feel a tumour. The abdomen may be retracted in this form of obstruction, especially if the jejunum is the part obstructed. The above facts are the chief points which indicate a gall-stone as the cause of acute intestinal obstruction. Gall-stones are really the only foreign bodies which are likely to give rise to acute obstruction; fæcal accumulation, collections of magnesia, phosphate, etc., cause constipation, but rarely death from acute obstruction. I have notes of an interesting case I saw in 1891, however, of a more acute nature, and it can be considered as a case of acute obstruction.

The patient was a boy æt. 11, who was admitted

Digitized by Google

under the care of Dr. Goodhart on December 15th, 1891. His bowels had been opened without any difficulty on December 10th. On the following evening he attempted to pass a motion, but found he could not do so. On the 12th, 14th, and 15th he made several further attempts, but without success, and each attempt was accompanied by a good deal of pain in the rectum. On making inquiries after his admission it was found that on December 9th he went for a walk near the docks and was present when a cart laden with cinnamon bark upset. He filled his pockets with bark, and on the way home ate a considerable amount of it. On admission he was suffering from great pain in the lower part of the abdomen and rectum. His pulse was 90, and his temperature

FIG. 1.—A. A gall-stone impacted in the ileum. B. A piece of liver, gall-bladder, and duodenum showing the cholecysto-duodenal fistula.

was 98·4°. His tongue was furred and the breath very foul. A rectal examination revealed the presence of a large hard mass, which felt like an enterolith. Purgatives and enemata were of no avail, in fact the nozzle of the Higginson's syringe could not be introduced for more than half an inch. An attempt was made to clear out the rectum, but it gave so much pain that an anæsthetic had to be administered, and then with a good deal of difficulty and with very free bleeding a hard, solid, mortar-like mass, which nearly filled a porringer of two pints capacity, was cleared out by means of a spoon. The mass removed was found to consist almost entirely of small chips of cinnamon bark. On the 18th he passed a normal motion, and on the 21st was discharged quite well.

I will now pass on to the consideration of the

Digitized by Google

second group of causes, viz. pathological conditions involving the gut itself, and the first sub-division I mentioned above was (*a*) *strictures becoming acutely obstructed.*

A stricture of the intestine usually causes chronic obstruction, but occasionally acute obstruction supervenes, a condition which, when it does occur, is nearly always brought about by some hard substance inside the gut blocking up the lumen at the seat of the stricture. I have separated these cases from the cases of obstruction by foreign bodies on account of the fact, that if it had not been for the stricture there would have been no obstruction, the foreign body, the actual cause of the obstruction, being much too small to block the lumen of a healthy piece of gut. A stricture of the gut may be defined as a narrowing of the lumen of the gut, the result of morbid changes in its wall. Strictures may be the result of cicatrisation after ulceration, *e. g.* tuberculous, dysenteric, syphilitic, and catarrhal ulceration (I do not believe that it ever occurs after typhoid fever), after the injuries which may follow strangulated hernia, and also after primary injury to the gut. The commonest cause of stricture, however, is carcinoma. The following case well illustrates this form of acute intestinal obstruction.

The patient was a woman æt. 61, who was admitted under the care of Mr. Lucas for a distended abdomen and constipation. She had been suffering for a year from troublesome constipation, and during the last few months had had six attacks of acute abdominal pain, vomiting, and constipation. For the nine days previous to the date of her admission she had suffered from complete obstruction, with vomiting and great distension. Soon after admission to the hospital laparotomy was performed, the colon was found to be very distended, and an artificial anus was made, the transverse colon being opened and attached to the abdominal parietes. She however gradually sank, and died on the following day. At the post-mortem examination, which I performed on the following day, a tight carcinomatous stricture of the descending colon one and a half inches above the sigmoid flexure was found, with a lumen which would only just admit a No. 12 catheter; the upper part of this stricture was completely blocked by a cherry stone.

I have here a specimen of carcinomatous stricture of the sigmoid flexure (Fig. 2) which is almost identical in appearance with the case I have just related to you. The stricture has been laid open, and shows a deep and narrow longitudinal ulcer, with raised warty edges infiltrated with a growth of

FIG. 2.—Carcinomatous stricture of the colon.

cylindrical-celled carcinoma. The muscular coat above the stricture is hypertrophied. The patient was a man aged forty-four, who was admitted under Dr. Habershon for intestinal obstruction of two weeks' duration, with occasional vomiting; signs and symptoms of peritonitis supervened, and five days after admission he died. At the post-mortem examination three pints of turbid serous fluid were found in the peritoneal cavity. Signs of acute intestinal obstruction might have arisen if the stricture had become obstructed by a hard foreign body.

Stricture of the small intestine occasionally

Digitized by Google

occurs, but it is extremely rare in comparison to stricture of the colon and rectum. I remember an extraordinary case of multiple tuberculous strictures of the small intestines in which occasional attacks of acute obstruction occurred.

The patient was a woman æt. 31, who was admitted on October 5th, 1894, under the care of Dr. Goodhart, for diarrhœa, anæmia, and general œdema. One of her relatives had died of phthisis. When she was twelve years of age she suffered from "mesenteric disease," She had suffered from chronic diarrhœa off and on from December, 1892, until the date of her admission. The day after her admission the bowels were opened ten times. This was followed by a period of constipation, and then again diarrhœa. When the diarrhœa was checked by astringents vomiting came on, and was associated with constipation; when the vomiting and constipation were treated there was a return of the diarrhœa. She died on November 22nd, 1894.

At the post-mortem examination, on opening the abdomen, numerous calcareous nodules and small masses, which were obviously healed tubercles, were seen, and also organised adhesions. The intestines were of small size throughout, and a number of strictures were found, the result of chronic tuberculous ulcers, and the lumen of the gut at the seat of some of the strictures was so small that it would only with difficulty admit the little finger.

Here is a specimen of tuberculous strictures of the ileum (Fig. 3), which is almost identical in appearance with the ileum found in the case I have just mentioned to you.

The above two cases illustrate the point I have already drawn attention to, viz. that the stricture itself does not give rise to the symptoms of acute intestinal obstruction. In the first case the acute symptoms were obviously due to the blocking of the lumen of the narrowed portion of the gut by the cherry stone; in the second case, as long as the motions were fluid there was no sign of obstruction, but as soon as the diarrhœa was checked signs of obstruction, viz. vomiting and constipation, supervened:

The diagnosis of such cases is not difficult, for usually there is some previous evidence of chronic obstruction, and when in such a case the symptoms become acute, it is nearly always due to the stenosed portion of the gut becoming blocked. Malignant strictures may, however, give rise to intussusception, and in this way acute symptoms may also arise.

(*b*) *Intussusception* or invagination, *i. e.* when a piece of gut slips into the portion immediately adjacent to it, is a common cause of acute intestinal obstruction. Brinton collected and analysed 500 cases of intestinal obstruction, and found that

FIG. 3.—Multiple tuberculous strictures of the ileum.

215 of them were due to intussusception; it is also by far the most frequent cause of intestinal obstruction in children, nearly 60 per cent. of the cases occurring in patients under ten years of age, and of these 25 per cent. during the first year.

Another point of interest is, that boys are much more liable to this form of intestinal obstruction than girls. The chief, if not the essen-

Digitized by Google

tial, factor in the causation of this disease is irregular peristalsis, and more particularly so if it is sudden, severe, and local. Irregular peristalsis may possibly act in this way:

The transverse muscular fibres contract, and in consequence elongate the portion of bowel affected (the entering layer) and force it into the portion of bowel below it (the receiving layer), whilst the longitudinal fibres of this last-mentioned portion of the gut may contract and draw it up over the entering part, so that there may be both a forcing down of the entering part and a drawing up of the receiving layer as a result of the irregular character of the peristalsis. That this drawing-up action of the lower part of the gut can take place may be shown by stimulating, a portion of exposed gut by means of the faradic current.

Irregular and violent peristalsis may be brought on by diarrhœa or by chronic constipation. It may follow sudden movements of the body. Fagge mentions two instances in which it came on suddenly in a child who was being jumped in its father's arms. It may also follow violent muscular exertion, and even injuries to the abdomen. The presence of undigested masses of food, the local irritation caused by intestinal worms, mucous polypi and other benign tumours, malignant growths, and even a diverticulum (invaginated) may be sufficient to bring about intussusception, the above-mentioned conditions causing irregular and severe peristalsis, the peristalsis being produced with the object of expelling the body from the part of the intestine in which it is situated. In the case of polypi attached to the mucous membrane or tumours involving the mucous membrane, they might be caught by the contraction of the gut immediately below it, and the part of the gut to which they were connected might be thus dragged down into the portion of gut below it.

Treves writes that in 50 per cent. of the cases no obvious cause can be found; he states that polypi are responsible for less than 5 per cent. of the cases.

I have here an interesting specimen, from the museum of Guy's Hospital, of a portion of the ileum, showing a short diverticulum which is inverted and which was found at the apex of an intussusception, and was thought to have been the cause of it. The diverticulum was situated 30 inches above the ileo-cæcal valve.

Invaginations may occur at any part of the intestinal tract. They may be classified thus:—(1) ileo-cæcal; (2) ileo-colic; (3) enteric; (4) colic; (5) rectal.

Of these the commonest variety is the ileo-cæcal (44 per cent.), in which form the ileo-cæcal valve forms the apex of the intussusception, the ileum the entering layer, the cæcum the middle layer, and the colon the receiving layer.

Fagge writes, "The special liability of this part of the bowel to the disease is probably the combined result of two different circumstances: one, that the axis of the large intestine is nearly at right angles with that of the small intestine; the other, that the cæcum is much more fixed, as well as much larger than the ileum."

I have here a typical specimen of the ileo-cæcal intussusception (Fig. 4). The invagination is about two inches long. The apex of the intussusception is the ileo-cæcal valve, and the colon forms the receiving layer. The end of the entering gut is indicated by the yellow rod, and close to it is seen the orifice of the appendix vermiformis, into which a red rod has been inserted. The specimen is from a child aged eight months, who died with acute intestinal obstruction. The duration of the illness was three days.

The rarest form is the ileo-colic (8 per cent), *i. e.* when the ileum is invaginated through the ileo-cæcal valve.

An intussusception is usually curved in shape, on account of the mesentery which is attached to the invaginated portion. As the intussusception increases in size through the entering and middle layers advancing at the expense of the outer layer, the mesenteric vessels become compressed, and finally strangulated, a condition of affairs which leads to congestion and œdema of the invaginated portion, and to actual hæmorrhage from the mucous membrane into the lumen of the gut below. Peritonitis may be set up and lead to adhesions between the entering and receiving layers, a condition which may hinder any further increase in the size of the intussusception, may prevent the possibility of its reduction, and as a result of the strangulation of the vessels the invaginated portion may slough off and be passed *per rectum*. If the peritoneal adhesions binding the entering to the receiving layer are firm, no general peritonitis, but recovery, may follow.

Digitized by Google

I have here a specimen of the cæcum with the whole of the ascending colon (Fig. 5), which was passed during life. The patient was a boy æt. 6, who was seized with abdominal pain and vomiting, and eleven days afterwards passed the piece of gut I am handing round to you. He made a complete recovery, and was seen forty years afterwards at the out-patient department, which he attended for indigestion.

FIG. 4.—Ileo-cæcal intussusception.

FIG. 5.—A cæcum and ascending colon which was passed during life.

The diagnosis is not as a rule difficult. The chief indications are a sudden onset, vomiting, spasmodic pain, abdominal tenderness, an elongated tumour in the course of the colon (in nearly 70 per cent. of the cases), and the passage of blood and mucous *per rectum*. A digital examination of the rectum should always be made, for in about 25 per cent. of the cases a tumour may be felt.

(*c*) *Volvulus* or twisting of the gut may be produced and cause obstruction in two distinct ways. In the commoner form there is a twisting of the gut about its longitudinal, or meso-colic, or mesenteric axis, so that at the point where the ends of the coil cross each other the lumen of the gut is obstructed, strangulation follows, and the affected portion of the intestine becomes enormously distended. In the rarer form two loops of intestine are involved, and are either doubled round each other or knotted together so as to produce a similar result. Volvulus may complicate hernia and other forms of obstruction, but I do not propose to consider these varieties in this paper.

The parts of the intestine which are most frequently affected are the loosely attached portions of the large intestine, the smaller intestine is, how-

Digitized by Google

ever, occasionally affected. Volvulus may be classified as follows:—(1) Volvulus of the sigmoid flexure: (2) volvulus of the cæcum; (3) volvulus of the small intestine.

(*c*) *Volvulus* of the sigmoid flexure is by far the commonest form, nearly 80 per cent. of the cases being of this variety. The following are the most important factors necessary for the production of this condition:—A long, narrow meso-colon and a long loose loop of colon, the ends of which are close together.

This abnormal state of the sigmoid flexure is most probably the result of chronic constipation, which would lead to constant over-loading of this part of the large intestine, would cause it to hang down into the pelvis, and so drag on the meso-colon and lengthen it by constantly putting it on the stretch, and in consequence would also approximate the two ends of the loop. The actual twisting might be brought about by some sudden or violent muscular movement, or by some unexpected change in position. In some of the cases the free end of the loop is found to be bound down by old peritoneal adhesion to some distant part of the peritoneal cavity, a condition which might also have helped to cause stretching and elongation of the meso-colon. The view that these abnormal conditions of the gut and meso-colon are congenital is disproved by the appearance of the intestine in young people, and also by the fact that volvulus is of rare occurrence before the age of thirty. I have already mentioned one important alteration in the condition of the strangulated loop, viz. distension; in addition there are usually extensive inflammatory changes, and the loop is full of fluid, conditions which led Dr. Bristowe to suggest that enteritis was the primary condition, and that the twisting was a secondary change and the result of the enteritis; but, as Dr. Fagge pointed out, this is disproved by the exact limitation of the inflammation to the part of the bowel strangulated. The twist may consist of one, two, or even three turns about its longitudinal axis.

In the other form, when two loops of intestine are involved, viz. of the sigmoid and of the small intestine, both loops are unusually long, freely movable, and with long mesenteric pedicles. The method of strangulation is best described in the words of Treves.

"The loop of small intestine falls in front of or across the pedicle of the sigmoid flexure; the flexure then winds itself around the axis formed by the lower coil; it passes upward in front of the loop of small intestine, and then moves backwards and downwards, so that the free end passes behind the pedicles of the two coils. In this way the abnormal sigmoid flexure forms a complete turn around the coil of the lesser intestine. Both segments of the bowels become strangulated, but the occlusion will be most severe in the axial loop."

In volvulus of the cæcum some congenital defect is almost invariably found; with the ascending colon there may be an abnormally long meso-colon, and the twist may occur about the meso-colic axis, or the cæcum may be bent or twisted about itself.

With regard to the small intestine, a loop may have an abnormally long mesentery, and may be twisted in its vertical mesenteric axis, or two loops of small intestine with abnormally long mesenteric attachments may be twisted or knotted about each other.

In September, 1900, a most remarkable and interesting case of volvulus of the ileum came under my care.* The patient was a young man æt. 21. He was admitted into Guy's Hospital on September 18th, for pain in the abdomen. On the 17th he was quite well until about an hour after his supper, which meal he finished by eating a large portion of damson pudding. At ten p.m. he was seized with severe pains in the hypogastric region, which were so bad that just after midnight he came to the Hospital and was admitted. On admission his pulse was 80, temperature 99°, and respiration 24. He was in great pain, which was paroxysmal in character. The lower part of his abdomen was rigid and tender. He was not considered by the house physician to be seriously ill. At eight p.m. on the 18th he passed a large mass of undigested damson skins, and with it about a pint or more of blood. After this he became very collapsed. I saw him at 9.15 p.m. for the first time and found him blanched, restless, and looking extremely ill. Blood in small quantities continued to be passed *per rectum*, but nothing abnormal could be felt *per rectum*. He gradually became worse, and died at 5.30 a.m. on September 20th. I made a post-

* 'Clin. Soc. Trans.,' 1901.

Digitized by Google

mortem examination, and found a volvulus of the ileum; I have the specimen here for you to see (Fig. 6); 100 cms. of the ileum, 10·5 cms. above the ileo-cæcal valve is involved; this portion of the gut at the time of the necropsy was of a dull, red colour, its peritoneum was covered with flakes of lymph,

FIG. 6.—Volvulus of the ileum. X = The point of torsion.

and some of the coils had a somewhat gangrenous appearance. The affected portion of the gut had a large mesentery which had become twisted one and a half complete turns in its longitudinal axis. I examined very carefully, but could find no sign of embolism or thrombosis of the mesenteric vessels.

The above case was not diagnosed during life. Such a profuse hæmorrhage is most unusual under such conditions. With reference to hæmorrhage occurring in intestinal obstruction Treves writes:—"I have met with two or three recorded instances where blood is said to have been passed in acute cases other than intussusception." No further mention of hæmorrhage is made. Its occurrence as I have mentioned above, is one of great rarity.

The diagnosis of volvulus is difficult, often a correct diagnosis is not made until the abdomen has been opened. The symptoms develop rapidly, vomiting occurs very early, is severe, and soon be-

Digitized by Google

comes stercoraceous. Enormous distension is seen, and it develops very quickly. There is intense pain and tenesmus. There is often a previous history of constipation.

(*To be concluded.*)

Errors in Laboratory Diagnosis.—Janeway calls attention to a number of errors which occur in laboratory clinical diagnosis. In urinary examination the most conspicuous error is failure to filter the urine, which makes a delicate test impossible. The most reliable tests are Heller's, the acetic acid, and ferrocyanide, heat and acid tests. As a general test Heller's is undoubtedly the most useful. No conclusion, however, can be drawn from the presence of albumin in the urine, excepting that it is not normal. The greatest significance of the negative test is in cardiac cases in which it gives evidence of absence of congestion of the kidney and makes the prognosis more hopeful. In the sugar test there are many serious errors especially with Fehling's solution. It is most useful for just one purpose, viz. to exclude the presence of sugar. The tests which give positive indications of the presence of sugar are the polariscope, fermentation and the phenyl-hydrazin reaction. The second one of these has some advantages in being useful also for quantitative determination. The phenyl-hydrazin test as ordinarily given is the most unsatisfactory, but the use of pure phenyl-hydrazin, 5 drops, glacial acetic acid, 10 drops, and saturated solution of sodium chloride, 1 c.c. to 10 c.c. of urine, boiling vigorously and letting cool slowly in the air has given him most excellent results. Sugar in the urine does not necessarily signify diabetes. The difficulties of clearing a mixed twenty-four hours' urine is considerable, and without this a fairly representative sample is hardly to be obtained. An excellent method where it cannot be had is to take equal parts of the water passed in the late afternoon before retiring and upon rising in the morning, but this will not suffice for careful quantitative methods. One of the commonest errors in connection with specific gravity is to conclude that the urine does not contain sugar, if it is below some arbitrary point, 1030 or 1020. He has often seen it with a specific gravity of 1015. Another mistake is failure to recognise the significance of the urine of constant low gravity and increased quantity. If albumin is absent such urine points strongly to contracted kidney.—*Journ. Amer. Med. Assoc.*, May 18, 1901.

FOUR CLINICAL LECTURES
ON
SOME AFFECTIONS OF THE KIDNEY.

Delivered at Westminster Hospital.
By A. H. TUBBY, M.S., F.R.C.S.

Lecture II.

Gentlemen,—The subject for to-day is renal calculus,—that is to say, stone in the kidney. Calculi in the kidney may be either uniform or lamellated. By uniform is meant a stone composed of one definite chemical substance, whereas in the lamellated form you find alternate layers of, it may be, uric acid, phosphates, and oxalates. It is important to recognise this point, because it is evident that drugs which will affect uric acid or urate of soda are absolutely useless in the case of oxalate of lime. This fact, of course, limits medical treatment. As to the composition of the calculi, the three most common varieties are uric acid, phosphate of lime, and oxalate of lime. It is comparatively easy to distinguish between them. The uric acid calculi are generally of small or medium size, though sometimes large; they are smooth on the surface, and are almost invariably of a light fawn colour. The oxalate of lime calculi are small or medium-sized, but occasionally large. They are known as mulberry calculi, because they are rough and very heavy, and they are dark brown or black in colour. Phosphate of lime calculi do not consist of the triple phosphate, but the dibasic phosphate. The triple phosphate is only found in ammoniacal urine, and it is found in the bladder. Phosphatic renal calculi are dibasic. They vary in size; they are not smooth; they are light in colour, and also extremely light in weight. With these ingredients there are often mingled urates of soda. You rarely find calculi in the form of xanthin which are yellow, and cystin which are green. One may say that the calculi which are of old standing are, as a rule, of the lamellated variety.

Another way of describing calculi is to speak of them as having a nucleus and a body. This nucleus varies. In a child or infant it is urate of ammonia. In adult life it is uric acid, and later in life it is oxalate of lime. As long as calculi are small and smooth, and are not causing irritation, they do not give rise to symptoms, but when they

Digitized by Google

move about they annoy and call attention to their presence. In other words, the symptoms of stone in the kidney arise from mechanical irritation and the blockage of the ureter which may be produced by the stone.

As to the *symptoms*, I dwell here upon what I call the preliminary stage of lithiasis. It is a condition which is recognised by all medical men, and with which the public themselves are fully acquainted, although they do not know it by the name of lithiasis. By it we mean a state of pain in the back, yellowness of the conjunctiva, drowsiness, headache, and constipation. All of these symptoms are due to the retention of products of decomposition of proteids. Many men suffer from that condition for a long time, and it may last for years. Eventually these people pass "gravel." Urates are often mistaken for gravel. When the urine cools, the urates are precipitated out. In time, however, these patients begin to pass true gravel,—that is to say, the cayenne pepper grains of uric acid; and you can often see these grains in a cloud of flocculent mucus. Or they may pass oxalate of lime or an excessive amount of urates. Patients with lithiasis are troubled by shooting pains in the loins, which run down towards the bladder, and such patients have acid urine, so that they are subject to slight attacks of balanitis. They have a sense of weight in the loins, which is aggravated by any slight indiscretion. Those who do not take sufficient exercise are also liable to gravel, and they get into a bad condition from it. They go to a medical man who prescribes certain things, but the evil only recurs if they do not observe certain rules. The next stage is the formation of calculus, together with the irritation which is produced by it. When a man has renal calculus, as a rule he has a definite train of symptoms. He has pain, but it is not of a constant nature, as in this patient here; it is fixed in the lumbar region, and it is of varying character. It may be of a dull, gnawing character, or of a pricking or stabbing kind, and if a calculus has formed such pain is increased by exercise, whereas if it is only gravel the pain is diminished by exercise: it is often relieved by rest and pressure. There is one striking point about these gravel cases, viz. that the pain is very much worse on rising in the morning. The next symptom of stone is the frequency of micturition, a symptom which is very constant. Patients will tell you that of late, since they have had the pain, they have had to get up twice in the night to pass water. It is important that you should recognise this, because frequency of micturition is a distinguishing point between gravel and true stone. Thirdly, these people suffer from flatulence, and they have distension of the colon. Why it is so is very difficult to say. Some think it is due to an inhibitory action upon the vaso-constrictor colic nerves. There are other theories, too, but no one seems to know. One thing is certain, and that is that the flatulence is very distressing.

Having elicited these symptoms you proceed to examine the urine, and you will find from time to time, but by no means always, a fair amount of blood if stone is present. The urine is smoky in character; but if the amount of blood is excessive, it may be of a bright red colour. The blood in this form of disease is diffused equally all through the urine; it is not found in clots in the same way as it is in malignant growth of the bladder. Next you will note that in the urine there are crystals of oxalate of lime or of uric acid, and in advanced cases you will also note pus. In examining urine for renal calculus it is also important that you should note the amount of urine which is passed daily and the amount of urea, for, in the absence of pus, these are the only guides we possess which will enable us to determine the condition of the kidneys, and whether the secreting substance is natural or not. A careful compilation of the amount of urine passed from day to day should be made.

I have spoken of hæmaturia. This is increased by exercise. The late Dr. Moxon, if he suspected calculus, sent the patient to walk round Finsbury Circus several times. By that means he secured better evidence of blood in renal calculus than he would otherwise have obtained. Another striking thing about hæmorrhage in the kidney from renal calculus is that although it is persistent in amount it does not cause anæmia, whereas in malignant disease the patient suffers from anæmia. There are certain fallacies with regard to hæmaturia which you must bear in mind. In a case of renal calculus there may be no blood in the urine; and for this reason, that there may be a stone blocking up the ureter, so that the affected kidney cannot pass its urine down the ureter, and

Digitized by Google

therefore there is no blood to be seen. Again, blood may disappear temporarily from the urine, or it may be associated with other conditions, such as tubercular kidney, granular kidney, Bright's disease, or growths.

Another, and a late sign, is the presence of pus cells in the urine. I want you to particularly note that if the pus proceeds from the kidney the urine is acid, whereas if it is from the bladder it is, as a rule, alkaline. Pus in the kidney, or from it, is usually associated with a large calculus, or with a very small movable stone.

Then there is tenderness on pressure in the lumbar region. In almost all cases where there is a stone present, there is distinct tenderness on pressure not only over the anterior aspect of the kidney, but over the posterior aspect. And if you give a rather sharp push with one or two fingers into the loin, the patient will at once call out and complain of a sharp stabbing pain. In speaking of the diagnosis of appendicitis from other conditions I alluded to this symptom of tenderness, and it is often difficult to diagnose pain arising from the appendix from pain due to the kidney. It has been pointed out, however, that in pain arising from the appendix, the anterior surface of the abdomen is more tender than the posterior, whereas in pain from the kidney the reverse is the case. That is a useful diagnostic sign.

Then these patients may have renal colic, and those who have seen patients with this condition will not readily forget it. They are suddenly seized with agonising pain in the loin, which radiates to the testicle, and the testicle becomes retracted. The pain comes on in paroxysms, and these paroxysms correspond to the peristaltic action of the ureter. There is rigidity of the abdominal muscles; a little blood comes away after the attacks, and perhaps the calculus also is expelled. There are collapse, vomiting, flatus, and rigidity of the abdominal muscles. These patients also have enlargement of the kidney, and may get hydronephrosis; you will understand how later on. This may go on to pyonephrosis, and finally there may be perinephritic abscess.

What becomes of the renal calculus? It may be passed. That event occurs more frequently than we in hospitals are apt to think. It is more usual for patients to pass it than for them to be operated upon for its removal. Chronic inflammation of the substance of the kidney and renal cirrhosis complicate renal calculus. The stone may be absolutely encysted in one of the calices of the pelvis. In such an event, unless the stone sets up acute inflammation about it, it gives rise to no further trouble. If a man retains a stone in the pelvis of his kidney for many years it sets up inflammation, which is known as pyelitis. This extends along the lymphatics of the kidney substance and causes numerous small abscesses. That condition is known as surgical kidney or suppurative nephritis. Other results of renal calculus are hydronephrosis, pyonephrosis, and perinephritis.

Now we come to the point at which it is important to ask one or two questions. First of all, is it possible to diagnose if the calculus be in the pelvis of the kidney or in the substance? That is an important question before operating. It is comparatively easy to find a calculus in the kidney substance, and when you begin to operate it is well to have a definite course marked out, and for that purpose it is better to know where the stone is. Unfortunately it is not possible to diagnose that with certainty, but you may make a shrewd guess. If there be excessive bladder irritability and constant pain in the testicle and much hæmorrhage, the stone is probably in the kidney substance.

There is another contingency which you must not forget, and that is that the second kidney may not be free from stone. It is very difficult to be certain, because it often happens that the same condition which gave rise to the stone in one kidney will cause it in the other. It is thus of the utmost importance that you should pull a kidney about as little as possible during operation.

You have to distinguish, if possible, renal calculus from gravel. I have said that gravel is the preliminary condition of stone. In some bad cases of gravel you find hæmaturia, but not in the majority. Other things being equal, hæmaturia is presumptive evidence of renal calculus. The important point is that these lumbar and testicular pains of gravel sometimes disappear by treatment and by suitable exercises, whereas such treatment has no effect upon stone except to make it worse. So you watch your patient carefully to see if suitable treatment for gravel causes a disappearance of the condition.

Diagnosis—It is difficult to distinguish stone in the kidney from tubercular kidney. In both there

Digitized by Google

are pain, frequent micturition, and hæmaturia. But if pus should appear in the urine in the early stage, that is in favour of tubercular kidney rather than of stone. On examining the urine from a case of tubercular kidney you may find tubercle bacilli. If you do not, and still suspect it to be of that nature, an injection may be made into the tissues of a rabbit, the animal being allowed to live for some time, and then a *post-mortem* examination made. In the urine of tubercular kidney, also, you will find caseous matter, and on boiling with caustic potash you will see the remains of connective tissue of the kidney. The next condition for which stone in the kidney has been mistaken is hydronephrosis, due to kinking of the ureter, with temporary blockage. This diagnosis can only be determined by palpation and operation.

Strange as it may seem, a large number of mistakes have been made between stone in the kidney and disease of the spine. I know of a case in which a spinal abscess opened into the pelvis of the kidney and discharged through the bladder, and you will thus understand that it is not so difficult for mistakes to occur. I have also known a spinal abscess to wholly surround a kidney with pus, and several parallel cases are described. You can only grasp the exact condition by carefully examining the spine, noting the presence or absence of rigidity, and so on. In malignant disease of the kidney you always have swelling in the lumbar region, which is not present in cases of stone in the kidney, and much hæmaturia and anæmia. The next question is the value of X rays in diagnosing stone. One may say that the dibasic phosphatic calculi are as a rule invisible when examined by X rays; uric acid calculi throw a light shadow; but oxalates, from the size of a cherry stone upwards, are shown distinctly in the radiograph.

Now is it possible to diagnose the size of the calculus, and also the part of the kidney it is in? A small calculus situated in a healthy kidney generally gives rise to two symptoms: (1) hæmaturia, (2) pain. There is neither pus nor enlargement. A large calculus which is situated in an abscess sac or inflamed pelvis gives rise to pus in the urine, tumour in the loin, rigidity of muscles over it, pain on pressure, and an evening rise of temperature. A small calculus which gives rise to hydronephrosis will cause repeated attacks of lumbar pain, and occasional excessive flow of urine. There is a swelling of variable size in the loin, which is painful on pressure. As to the calculus being in the pelvis or in the kidney substance, you must remember what I have said; if it is in the kidney substance there will be much hæmaturia, and excessive testicular pain.

We now come to *prognosis*. The prognosis here is comforting. One may say that of three patients who have stone in the kidney it is only one that dies of it. But there is this to discount that prognosis; that although two of three patients do not die of it, they have an extremely bad time.

The *treatment* may be either medical or surgical. It is evident from what I have said that medical treatment is of value in the condition known as gravel. I need not go into this point minutely, but by giving citrate of potash, and telling the patient to eat little meat, to take copious draughts of water, and to lead a healthy outdoor life, the gravel often disappears very rapidly. Sometimes salicylate of soda is given, and thirty grains of tincture of hyoscyamus and water to one ounce three times a day will be found beneficial. If the calculus is of a simple nature, that is to say, not lamellated and composed of uric acid, it is claimed that the continued administration of citrate of potash may cause such a calculus to dissolve. A scientific-minded physician took a stone which was composed of uric acid and weighed it; then he put it beneath a jet of fluid in which a certain proportion of citrate of potash was placed, and he published the result of his researches, showing that the stone lost so many grains per hour, forgetting that it is hardly possible for that to happen in the body. Vital chemistry is a very different matter from test-tube chemistry.

After a long time, and after the patient has suffered greatly, should the case be operated upon? There are two operations which are open to you, nephrolithotomy or nephrectomy. Of course nephrectomy is a last resort, and it is only done when the kidney is disorganised, and this operation comes last in the purview of renal calculus. Many of you have seen the operation of exploring the kidney, and you are aware how it is done. The incision is made at the right margin of the erector spinæ, half an inch below the last rib, extending downwards and forwards five inches. The various muscles are divided, and you make

Digitized by Google

sure of the position of the colon. You then gently tear through the perinephritic fat and come upon the kidney. The important part of the operation is a very careful search of the whole of the kidney, and it is conducted on well-defined lines. Firstly, you take a properly shaped needle and stab the kidney at twelve or fifteen points, and in all directions. You must take care that you do not stab more than two and a half inches in depth, and not so much as this at the thinner parts of the kidney. You may sometimes come across a stone in this way, but there may be considerable hæmorrhage, which produces trouble. If this method should fail the kidney should be palpated between the finger and the thumb, and at any suspicious point where it is harder than usual the needle should be passed in to see if you can strike the stone. If you find no evidence of stone by this means you should open the pelvis of the kidney, put your finger in and feel all round, and then explore with a sound; but if there be no result it is permissible to make an incision along the posterior wall of the kidney, split the kidney substance for some distance, and explore again with the finger. If you find the stone the next difficulty may be to extract it. For this purpose you need a proper pair of forceps, and if the stone be very large you will need a lithotomy crushing apparatus to reduce it to fragments, and then remove it. In at least one fifth of the cases which are operated on for stone no stone is found, notwithstanding that the patient may present all the signs of this trouble. But you are justified in operating when the symptoms have persisted for a long time. If there has been much hæmaturia, and the patient's life is burdensome to him, you should operate, and you should also operate if there is pus in the urine. The after treatment of these cases is a matter of mere surgical commonsense.

Lichen planus treated as a Syphilide with Mercury.—Dr. T. G. Lusk considers that mercury in doses of one twelfth of a grain bichloride, half a grain protoiodide, and one sixth of a grain biniodide, is the best treatment for lichen planus. He now firmly believes that the affection is a syphilide, the excellent results from mercury affording the clear and only corroboration of his opinion.—*Journ. Cutaneous and Genito-Urinary Diseases*, May, 1901.

WITH MR. JONATHAN HUTCHINSON AT THE MEDICAL GRADUATES COLLEGE AND POLYCLINIC.

Lichen Spinulosus.

Here is a boy who exhibits lichen pilaris or lichen spinulosus, and he has had the condition six months. His family history is distinctly tubercular, that is to say, his grandfather and grandmother on the mother's side died of consumption, and the mother has lost three sisters and four brothers. That is a very strong history. The mother is living, and appears to be perfectly healthy. There are many points which are to be noted about the case. By lichen we of course mean some eruption beginning around the hair follicles. Possibly in some cases the eruption has some relation to the sweat-glands instead of the sebaceous glands, and consequently there are little papules. "Spinulosus" is the term applied when there are these little spines, which consist, I believe, of consolidated sebaceous material. In some cases they feel rough, like a nutmeg grater, without there being any distinct spines. This boy has the condition very marked in the popliteal space. There is thus a history of what is called lichen scrofulosorum, and I may say that is generally accepted as a miliary tuberculosis of the skin. This is one form in which tuberculosis of the skin occurs. We must be a little careful in our diagnosis, because it might be a case allied not to lichen scrofulosorum, but more to ichthyotic conditions, meaning by that a congenital dryness and imperfection in the development of the skin. This condition of lichen scrofulosorum very rarely occurs in the subjects of phthisis, but affects those who have near relatives suffering from tuberculosis. The skin in this boy tends to be slightly "spinous" in all directions. If you get a side view of the neck you can make the little "spines" out very well. It would be interesting to get microscopic sections of the "spines" to see if the material composing them is sebaceous. I believe that I was the first to apply the name spinulosus to the condition. I had a wax cast made of a beautiful specimen at the London Hospital, but the "spines" were larger than they are in this case. The question is whether this is a congenital imperfection in

Digitized by Google

the formation of the skin, or whether it is a tubercular affection. As a rule we have not associated the spinulosus form of lichen with lichen scrofulosorum; but we are now enlarging the domain of lichen scrofulosorum very much, believing that the tubercle bacillus accounts for very many forms of dermatitis which previously we have not so associated. In this boy we have demonstrated that the skin is rough everywhere, but we do not recognise any particular patches. There is no evidence that the boy had any pre-existing disease; he seems not to have been ichthyotic, the skin having been perfectly soft before this came on. There was no evidence of any skin trouble until six months ago, and then it began in the neck, which is the usual situation for the condition. That is very interesting evidence against the idea that it is a congenital condition in this case. His brother has been seen, and possesses a very soft skin. The next positions to be affected were the arms, then the popliteal space, and now the condition has become diffuse and general. There is a general diffuse dermatitis which has occurred in connection with this tubercular invasion of the orifices of the hair follicles, and this is attended in some of them with little points of sebaceous matter, producing a sort of spine. I do not think they are papillary growths in any case, but I think the material consists largely of shed epithelium from the hair follicles. There are hairs growing in various parts. But for the history which is given we might well have supposed that the patient had a congenitally dry skin, because the distribution is so diffuse. In this case there is very little disturbance of health. In some cases of lichen scrofulosorum the tubercle bacillus has been identified with the disease. The skin diseases which we recognise as being associated with the tubercle bacillus are very numerous. I show you a drawing of a case of lichen scrofulosorum which was exhibited at the Dermatological Congress in London. There are minute brown lichen spots aggregated together in patches, not abruptly circumscribed, because there is a little shading off of their edges. I show you also Hebra's original drawing, and he described it as occurring in patches, but you will see there is a good deal on the surface of this skin which is not strictly in patches. Lichen pilaris is the term given to those cases in which there is evidently a retarded growth of hair in the follicle. If lichen pilaris is to include all diseases round hair follicles, that is the definition of lichen, and we do not want the term pilaris at all. Many of the names of diseases will have to be overhauled if there is a chance of simplifying nomenclature. Here is a drawing of a case of Kaposi's, a condition to which Kaposi now applies the term lichen scrofulosus. We enlarge the area of the disease to those cases where the follicles are inflamed, and we must allow for a certain margin of variation, understanding that the essential condition of lichen scrofulosorum is tuberculosis of the skin manifesting itself in a general irritation about the hair follicles, with overformation of epidermis about them. I show you a reproduction from a portrait, where the condition is abruptly marked in patches, and there is a very great concretion of epidermis around the edges. Here it is shown that the patches cover the fingers and face, places in which we formerly thought it never occurred. Next I show you a very interesting drawing of Dr. Crocker's, in which the disease does not occur in patches, but is scattered over the whole surface, and comes to the very ends of the limbs. There is an affection of the skin deserving the name general miliary tuberculosis of the skin, which manifests itself by the production of these lichen papules, and it is very common in scrofulous children. In a large number of instances cod-liver oil and the use of tonics has effected a cure. I show you next the portrait of a boy who I repeatedly showed, and his brother also. His father died of lung disease, though I could not get an admission that it was tubercular. This little boy had a brother with enlarged glands, and I had him under my care for the glands at the same time as I had the boy under treatment for the patches. It is important to recognise that we can have a tubercular affection of the skin taking place in this peculiar manner, not producing ulcerations, or any remarkable failure in health, but causing enlargements of hair follicles; and which is infective, though we do not know how; but probably by spreading over the surface of the skin by rubbing and scratching. Or it may be that there is some infection by means of the blood. In nearly every case there is some tuberculous trouble in the family, and they themselves have at least a suspicion of tuberculosis before the skin affection commences; some spit blood, others have tubercular glands, others have lupus.

Digitized by Google

The worst case I have seen was a patient with lichen scrofulosorum who had lupus vulgaris, and in whom there was a strong family history of tuberculosis.

Enlargement of the Epididymis.

Here is a young married man with an enlargement of the lower part of the epididymis, and indeed of almost the whole of the epididymis, the testicle itself being quite free from enlargement, and quite soft and healthy. The epididymis is almost as large as the testis, and there is considerable induration about it. It is hard enough to be tubercular disease, and it is sufficiently circumscribed and definite. It does not extend high up in the testis, and does not implicate the cord. There is no material enlargement of the vas deferens. One can move the body of the testis without hurting the patient. The lump is isolated, and about the size of a hazel-nut. The history is that it occurred only a few days ago, but of course that history may be misleading, inasmuch as, since it gives him no pain, it may have been there without having been noticed. I should recommend that he be put upon tonics and small doses of mercury, and told to wear a suspensory bandage. At first he had pain on the left side. He is the father of one child. It is not in the least like gonorrhœal orchitis, and the patient says he has never had anything in the nature of gonorrhœa. There is no apparent implication of the veins. It is in the lower part of the epididymis that tubercle generally begins in the generative organs. The treatment I have recommended is intended to produce absorption. Of course he should also have a liberal dietary.

Tabes.

Here is a man æt. 53, married, the father of three children, aged eighteen, sixteen, and four. About twenty years ago he began to be troubled with general neuralgia; in a few years it became more severe, and treatment appeared to produce no benefit. He brings a list of the various remedies which have been given to him, but they have been entirely useless. About four years ago he noticed he was rapidly losing the power of writing. Sir William Gowers has seen the patient, and confirms the opinion that it is a case of tabes. He ordered the patient complete rest at the seaside, and during that time he did not attempt to write. After a month there he returned to business and kept at his work two and a half years. But he again had to give up, as his fingers and hands and arms were not capable of much continuous work. Ten years ago he began to use antipyrin to relieve the severe pain. Two years ago he commenced using antifebrin, and he takes twenty-grain doses three or four times a day. He thinks antifebrin has done him the most good; and he says he could take that on an empty stomach, but not antipyrin. He says his doctor persuaded him not to take such large doses. He says he has sometimes taken four doses of twenty grains each in one day. There is loss of the knee-jerk, and it has been lost for some time. He has the severe lightning pains, which he says seem to go right down his bones. He has had gonorrhœa, but believes he has never had syphilis. He does not remember having any sore. He has had two attacks of gonorrhœa, which lasted a long time, because he was travelling. There is nothing else to corroborate the suspicion of syphilis. He says he cannot now raise a cup of tea or coffee to his lips. The arm is very numb, and yet it is very sensitive to neuralgic pain. He does not get pains in the head. The left eye has lost the power of accommodation, and the left pupil is larger than the right. There is no joint disease and no swelling. For a little time, about six years ago, he lost sensation in the soles of his feet, and he could not walk at a greater pace than two miles an hour. But that passed off in two months without treatment. It is very difficult for him to walk at all in the dark, but in the daytime he can walk one to one and a half mile slowly. It appears to be a case of tabes of very long duration, in which the characteristic pains have been the most prominent symptom; he has gone through twenty years retaining fair general health. Another peculiar feature, as far as my experience goes, is the severe implication of his upper extremities. His sphincters appear to be rather troublesome, as he says the urine dribbles a little, and sometimes he involuntarily passes his water at night-time in the bed. His bowels are rather constipated, but he can hold his motions, and has never had accidents in that respect. It is well to always remember to examine the sphincter ani in such cases; sometimes you may find that it does not contract on the finger at all.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 451. WEDNESDAY, JUNE 19, 1901. Vol. XVIII. No. 9.

CONTENTS.

PAGE

*A Clinical Lecture on Hydrocele. By A. MARMADUKE SHEILD, M.B., F.R.C.S. 129

*Some Remarks on the Causation and Diagnosis of Acute Intestinal Obstruction. By J. H. BRYANT, M.D., F.R.C.P. (*concluded*) 136

Diagnosis in Wry-neck. C. B. KEETLEY, F.R.C.S. 144

Erythromelalgia and Arsenic. T. N. KELYNACK, M.D., M.R.C.P., and W. KIRKBY, F.L.S. 144

*Specially reported for the Clinical Journal. Revised by the Author.

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE ON HYDROCELE.

Delivered at St. George's Hospital.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I propose to lecture to you to-day on some of the principal points concerning the pathology, diagnosis, and treatment of hydrocele of the tunica vaginalis testis. The subject is a large one, and you must supplement the brief remarks I have time to make by your own observations and reading. I have selected the subject because there has recently been in the Fitzwilliam Ward a man with a large old hydrocele, upon which the method of radical cure was carried out by operation, as the injection treatment had on two occasions failed.

Hydroceles form a large proportion of all scrotal swellings, and of the cases at surgical examinations. A good deal of difficulty is often experienced in their recognition by students who do not consider how much can be learnt in the diagnosis of such affections by the appearance to the naked eye, and other common-sense methods of examination.

But before discussing the diagnosis of hydrocele, we may say a word about its causation. This is summed up at once by declaring that in a large number of cases of hydrocele no certain cause can be assigned for the disease. The books will tell you that hydrocele is due to a loss of the balance between the absorption and secretion of fluid in the tunica vaginalis. This is the stock answer given at examinations. To my thinking it is only a convenient way of expressing ignorance. The fact remains that in a considerable number of cases of hydrocele the sac of the tunica vaginalis is distended with fluid without any rational cause being assignable. A very suggestive point is as follows: A certain number of cases of hydrocele are associated with definite disorders of the testicle. Thus, in syphilis of the testicle, concomitant hydrocele is familiar. In tubercle of the testicle, malignant disease of the testicle, chronic orchitis and epidi-

Digitized by Google

dymitis due to catheterization, hydrocele is also found, though far more rarely. Thus a certain number of cases of hydrocele are undoubtedly caused by the spread of inflammation to the tunica vaginalis from the testis or rather from the epididymis. This suggests that hydrocele is usually inflammatory in origin. I need hardly point out to you how important this is in the matter of treatment, for the method of curing hydroceles dependent upon syphilis, or tubercle of the testis, is largely bound up in the treatment of the testicular affection which causes and underlies the hydrocele. In some hydroceles a small pedunculated body is found near the head of the epididymis. This is a fœtal relic, and I have found it in the tunica, when no hydrocele was present. I have often wondered whether the rubbing of this substance against the serous surface might produce excess of secretion. The analogy of serous exudation in the knee, due to the rubbing of a pedunculated body from the edge of the cartilage, seems very close.

Next we will talk about the symptoms of the ordinary hydrocele of the tunica vaginalis, such as you often meet with in the out-patient department of a hospital or in private practice. The symptoms of this disease are really exceedingly simple. First of all, the shape of the swelling at once tells you that it is a distended tunica vaginalis. I cannot too much impress upon you to use your eyes in the diagnosis of scrotal tumours and swellings. Here I show you a drawing of a typical ordinary vaginal hydrocele. The swelling is pear-shaped and the neck of it is narrow, so that the cord can be distinguished above the swelling by the pressure of the thumb and finger, showing that no hernia exists, and that it is definitely isolated from the abdominal cavity. Large hydroceles have often a slight constriction about their centre, "hour-glass contraction," which is also very characteristic. There is no impulse on coughing, the swelling cannot be reduced into the belly, and it is very generally translucent. And here, I may remark, you should lose no opportunity of practising the translucency of a hydrocele before you present yourselves in the examination room. First learn to make the swelling tense by pressure with the hand behind it, and then, by "shading" it with the other hand, translucency is readily produced. A strong bright light, like an electric lamp, is most useful. But the method requires practice, and practice alone will make perfect. In the vast majority of cases of ordinary hydrocele the position of the testicle is what I have shown here in a diagramatic manner; namely, behind the collection of fluid. As a rule the patient is able to distinguish testicular sensation when this part of the swelling is squeezed. I advise you never to carry out such an examination, for you can rarely depend upon patients' statements as to their sensations, and a careful examination by transmitted light will usually show you the position of the testis by the opacity produced. In a large hydrocele, moreover, the volume of the fluid is so great that it is generally impossible to feel the testis at all, still less to squeeze it. You may recollect, then, that as a rule the testicle is behind in a common hydrocele, so when a trocar is entered in front, it is clear of the organ. But there are exceptions to every rule. In a few instances of hydrocele the reverse is the case, the testicle being in front of the hydrocele. This unusual circumstance may be due to malposition of the testicle, the epididymis being in front instead of behind. It may also be caused by the "anchoring" of the testis to the front of the sac by inflammatory adhesions. Thus this condition is found in old hydroceles, which have been tapped many times, and perhaps fruitlessly injected.

The fluid which is contained in hydroceles is highly albuminous, and is generally of a pale straw colour. In some cases of old hydroceles, which have existed for many years, the fluid becomes more opaque and mixed with cholesterin, the colour of old ale, which may greatly mar the certainty of the "translucency" test. Hydroceles associated with sarcomatous testis may contain bloody fluid. It would be more correct to call them hæmatoceles, except that the serum is often in great excess over the blood mixed with it.

Lastly, the fluid in a hydrocele is as a great rarity chylous. I have never seen a case of chylous hydrocele originating in this country, but I have seen one which came from abroad, and the presence of filaria in the lymphatics was suspected by experts.

All hydroceles are not translucent. Those which have existed for a great many years, and have perhaps been tapped many times, and have become chronically inflamed, are notably not transparent. The sac in such cases, from repeated inflammatory attacks, may attain to a great thick-

Digitized by Google

ness, as you will readily understand. This diagram represents an ancient hydrocele with a thick sac full of inflammatory lymph, containing particles of cholesterin. Such a swelling will be non-translucent, and the diagnosis may be puzzling enough, unless you obtain a clear history, of long duration, and frequent tappings. 'Histories' are very deceptive, and sometimes these cases can only be cleared up by exploratory incision. How, then, are you to certainly diagnose these elastic opaque scrotal swellings? The only way of certainly diagnosing hydrocele with thick walls is by an exploratory incision. It is impossible, for instance, to distinguish a hydrocele with thick walls from an old hæmatocele except by cutting into it; you have little or nothing to guide you except the history so apt to be fallacious. A swelling may be a hydrocele at one tapping, a hæmatocele at the next, the trocar having pierced a vein, which slowly oozes into the cavity. In these opaque swellings too, the position of the testis is very problematical. There are frequently inflammatory adhesions, which displace the organ. Hence the incision is safer than the trocar, for the latter may pierce the testis, and lead you to the erroneous conclusion that you have a solid tumour to deal with.

Confusion between ordinary vaginal hydrocele and soft sarcoma of the testicle ought, in reality, never to occur. Soft sarcomatous growth of the testicle is, on the whole, rare. Hydrocele is very common. Soft sarcomatous growth of the testis is non-translucent; it is heavy when lifted up on the hand. It feels solid in some parts, fluctuating in others. The scrotal veins are much enlarged, and the cord above is thickened. Of course an exploratory incision immediately clears the matter up, because instead of opening into a cavity containing fluid, you come upon a vascular brain-like tumour. Nevertheless confusion between these affections is possible. You may reflect that non-translucent hydrocele will have existed for years; soft sarcomatous growth of the testis will probably have existed for months, and will have progressively increased. Exploratory incision may be the only means by which the matter can be certainly cleared up. I warn you not to take into account the appearance of the patient. Large malignant growths of the testis may be found in persons who have the appearance of perfect health. Confusion between these two affections is very disastrous, both for the patient, and your own reputation.

I have said very little about the diagnosis of ordinary common hydrocele from hernia. Ability to feel the elements of the cord above the swelling, is the main differential symptom on which to rely. But I will mention as I am dealing with the subject that the shape of the neck of a hernia is different from that of hydrocele. In hydrocele the swelling is definitely cut off from the abdomen at the external abdominal ring; in hernia there is a wide neck which passes up into the abdomen distending the inguinal canal, so you can see at a glance the distinction between the two conditions.

The treatment of hydrocele is very variable. In a large number of instances, especially in private practice, you will find that the patients will be satisfied with the swelling being tapped at certain periods. This is termed the palliative treatment of hydrocele, namely, performing paracentesis when the swelling gets inconvenient from its bulk. This operation should be done with an exceedingly fine and sharp trocar and cannula. The patients will appreciate the use of a fine instrument. The hydrocele trocars sold in the shops are often too large for use by a sensible and humane surgeon. The skin of the scrotum should be carefully cleansed, and the instrument boiled before and after using. The sac should be made very tense by the pressure of the left hand before the trocar is introduced, and a spot should be selected free from veins. As I have said, you will find a large number of patients affected with hydrocele will be content with this palliative tapping, and this is especially true in elderly men engaged in business, or occupations which preclude them "laying up." Such patients are acutely critical, and if any bungling takes place in the little operation, they will not visit the surgeon who is guilty a second time! Now, reflect, you would not attempt radical cure of hydrocele which was dependent upon disease of the testicle. Under such circumstances it would be very unwise to undertake, for instance, the injection method. The diseased testis would be left, and perhaps the mischief in it aggravated by the irritant fluid. Tapping is often employed in hydrocele associated with testicular disease, and

Digitized by Google

indeed is sometimes essential in diagnosis. Not until the fluid be withdrawn can the surgeon properly feel the enlarged testis, and estimate its nature.

The next class of case where you would not attempt the injection cure is where there is a very thick sac. If you were to inject iodine into a hydrocele with very thick walls you would only make matters worse. Again, radical cure by injection should never be attempted where there is communication between the sac and the abdominal cavity. Such hydroceles are found in young infants and boys (congenital hydrocele), but many hydroceles in the very young are quite ordinary, and do not communicate with the belly; this fact students seldom appreciate.

One of the objections to radical cure by injection of iodine is its uncertainty, It has been computed by some authorities that 20 or 25 per cent. of the cases of hydrocele treated by injection are not cured. Another objection to injection, especially with iodine, is that the epididymis is generally inflamed by it, and so you get for weeks after the injection a painful indolent swelling of the testis and epididymis, which has been set up by the injection. The injection is very painful when the iodine enters the tunica vaginalis. Patients have been known to faint not infrequently. Still injection of iodine is a remedy which is practised largely, especially in hot countries like India, where hydroceles are very common and very large, and you should know how to do it.

Firstly, your trocar and cannula should be rather larger than for ordinary tapping. You should see that the cannula and syringe fit properly, so that you may rely on throwing the iodine into the tunica vaginalis without a hitch. You should draw off all the hydrocele fluid; do not leave part of it in the sac. A very common error is to leave several drachms of hydrocele fluid in the sac, and thus when the tincture of iodine is thrown in it gets diluted. I believe that many of the instances of "recurrence" after injection may be explained by some faulty procedure on the part of the operator. You throw into the cavity of the sac one to two drachms of the Edinburgh tincture of iodine, which is rather stronger than the usual preparation. The next step is to shake the sac well, so that the iodine is diffused all over it. I have told you how acutely painful this may be, and it is wiser to perform the operation with the patient lying down on a sofa. Some surgeons make a practice of previously throwing into the sac of the tunica vaginalis a 2 per cent. solution of cocaine. Not more than a quarter of a grain should be introduced. This certainly diminishes the smarting caused by the iodine. When you see the patient the day following the injection you will find the scrotum swollen and red, and the epididymis inflamed and tender. The patient will sometimes complain of very great discomfort, so that he has to be kept in bed for a few days, though this is unusual. As a rule there is more or less epididymitis, and a chronic indolent swelling results, which only slowly subsides.

An injection which is perhaps becoming more popular than iodine is carbolic acid. This is at least as likely to cure as iodine, and it is very much less painful. You take a mixture of equal parts of liquefied carbolic acid and glycerine, and, having emptied the hydrocele, you throw into the sac 40m to a drachm of this solution. This was the method I adopted in the out-patient department of this hospital for several years, and I have every reason to be satisfied with it. We had ten consecutive cases of cure, so far as I could ascertain. There were other cases "lost sight of,"—so common in hospital practice. The scrotum is shaken after injection, so that the whole sac comes into contact with the remedy, and a cure is generally the result. Carbolic acid does not, in my experience, inflame the epididymis to the extent that iodine does, neither does it cause such severe pain, so I think it is a preferable preparation. There are various other fluids recommended for injection into hydroceles. All of them, however, are inferior to iodine and carbolic acid, and carbolic acid is, I believe, superior to any. Before leaving the subject of the injection treatment, I may mention that the Indian medical officers especially have had good success with it, and that in certain cases it may with advantage be tried. For large old thick hydroceles I doubt its advantage.

The method of treatment which most modern surgeons perhaps advise for hydrocele is the radical cure by operation. It is an excellent proceeding in boys or young men who have to enter the services of the army and navy. If the radical cure by excision of the sac is performed, it necessitates resting in bed or on the sofa for

Digitized by Google

upwards of a fortnight or three weeks; but it is a method I always like to adopt, because of its certainty to cure the patient. The operation is done in various ways. The first is the simple incision method. The hydrocele is incised, and the interior of the sac is lightly rubbed over with carbolic acid with a strength of one in ten; a drainage-tube is then put in, and the incision sewn up. This is a good method for small hydroceles in boys. I do not care for it when the sac is large.

Then there is the method of excision of the sac, which is the course I myself prefer, as being the most certain. The sac of the hydrocele is exposed, and a considerable portion of the parietal layer of the tunica vaginalis is cut away with sharp-pointed scissors. The remaining portion, which covers the front of the testis, is lightly touched over with one in ten carbolic acid, or with a solution of nitrate of silver, twenty grains to the ounce, a drainage-tube is put in, and the parts sewn together. The tube is withdrawn in twenty-four hours, and the part carefully dressed and elevated. A certain amount of inflammatory epididymitis will ensue. Especial care must be taken in replacing the organ in the scrotum that the cord is not twisted. This is easy enough to do, and it may lead to very unpleasant after-consequences. In both these procedures the strictest asepsis must be maintained in the dressing and elevation of the part must be carried out carefully. An important precaution is to be on your guard against secondary hæmorrhage. You know that in a big hydrocele the little vessels are very much stretched by the pressure of the fluid, and are diminished in calibre. As soon as the fluid is let out the vessels become engorged. Sometimes a little artery or vein will bleed very persistently into the loose scrotal tissues in these cases. You must patiently tie every single bleeding point before you unite the wound, and send the patient back to bed, using ligatures of the finest catgut. If this is not done you may have troublesome secondary hæmorrhage into the scrotum, which becomes "blown up" and bluish in colour. Quite lately another operation has been practised, namely, inversion of the tunica. After incising, the sac is turned inside out, so that its serous surface becomes external. The results are said to be excellent. It is an operation I have not yet personally tried, and in the case of a large or thick sac I should think the excision method is preferable.

Such are the principal modes of carrying out the radical cure of an ordinary hydrocele of the tunica vaginalis.

I must now say a word about a very important practical matter, namely the treatment of hydroceles in little children. Male infants sometimes get hydroceles like their seniors, and the greatest alarm is manifested by the anxious female relatives. I fear what you see in the out-patient department gives you little idea of the anxiety and trouble manifested about a simple hydrocele in the first-born of an important family. Remember that the symptoms of hydrocele in a baby are exactly the same as those in the adult. You observe a little pear-shaped swelling, which is readily translucent, light showing through it as a red glare. One of the first things you have to determine is whether the swelling is a congenital hydrocele, and if the fluid can be reduced into the abdominal cavity. Having ascertained this is not the case, and that the swelling is an ordinary common hydrocele, how are you to treat it? I want to dwell upon this part of the subject, because I find medical students generally have an idea that the hydrocele of a baby boy can be treated in the same way as that of an adult. The answer one generally gets in examinations to this question is, that the hydrocele of a baby may be injected with carbolic acid or iodine. Now if you inject iodine or carbolic acid into the hydrocele of a little child, the scrotum will inflame and may possibly slough, and the life of the infant may be endangered. This is not a fanciful danger. The mistake has been made in practice, and I know of one most unfortunate case where great trouble ensued both to the little patient, and the reputation of the doctor!

How, then, are you to treat these swellings in the young? In the first place, the ordinary hydrocele of infants will not infrequently go away if it is left alone. This will seldom satisfy the parents; they demand some active treatment. The first measure to adopt is to paint the outside of the scrotum persistently with a slightly irritating lotion, such as chloride of ammonium. I use a strong solution, a saturated solution of the drug in rectified spirit and water. I am under the impression that this remedy really cures very few of these cases, but it is harmless. I have seen it succeed,

Digitized by Google

but I have been uncertain whether the hydrocele would not have subsided without it.

The next method is acupuncture. The scrotum is made tense, and a fine sharp needle is passed into the hydrocele in various situations; the fluid exudes in drops, and passes into the cellular tissue. This method is advocated in the text-books. I may tell you it is not a certain cure. I have for a number of years practised this operation, and always with success until last year, when I had two cases in which the fluid returned, much to the disappointment of the parents. It shows us how increased experience is apt to falsify any one "certain" remedy or panacea for disease. Therefore recollect that acupuncture, though a good method, may sometimes fail you. Were I called in to-morrow to a large hydrocele in an infant, especially if it had resisted other treatment, I should incise it. You have only to let out the fluid and rub the interior of the sac with a solution of nitrate of silver, 10 grains to the ounce, or 1 in 10 carbolic acid. This may be done by dipping the end of a probe into the "irritant" and passing it lightly over the surface of the tunica vaginalis. A small piece of gauze is inserted for the first twenty-four hours. If the sac is very large you can excise a portion of the parietal layer, just as in the adult. The most careful asepsis must be maintained. This is practically very difficult. I employ soaking in a warm boric bath very constantly in these cases. There is another point about hydroceles in infants; it is as follows:—In certain hydroceles in little babies the sac goes a long way up into the inguinal canal, distending it and simulating hernia. This is due to failure of obliteration of the lower part of the funicular process. This variety is called infantile hydrocele. A mere septum may separate the fluid from the abdominal cavity, and careful examination is needful for you to assure yourself that the rupture is not really of the congenital variety, and that the fluid cannot be pressed slowly into the belly.

So much for ordinary common hydroceles in adults and in children, and their radical cure. Now we must go on to say a word or two about a form of hydrocele which occurs generally in adults, so-called encysted hydrocele of the testis, or more properly the epididymis. You see from the diagram that the testis is below, and if the hydrocele is of very large size, the testis may be altogether in front and below, the scrotum presenting a peculiar appearance. These encysted hydroceles of the epididymis are said to have their origin in dilatation of some of the fœtal tubules which are found in that neighbourhood. The cysts do not, as a rule, attain to large dimensions. You find them about the size of a hazelnut or a walnut. They are tightly distended with fluid, and often mistaken by students for solid swellings; indeed, the sensation to the hand is exactly like a double testicle. Whenever you find what seems to be a double testicle on one side, you may be sure the case will turn out to be an encysted hydrocele; and when you manage the light properly, you will be able to see that the supposed solid tumour is translucent. These remarkable swellings contain perfectly clear fluid, which is non-albuminous, and a large amount of phosphates and carbonates. On the addition of nitric acid effervescence occurs. But inasmuch as a seminal tubule frequently ruptures into them, they become full of spermatozoa, which may give them a milky appearance and spoil their translucency. Then the fluid, of course, becomes albuminous. The walls of these cysts may also be thickened, and this prevents translucency, and the exploratory incision may be needful to establish a certain diagnosis.

They are treated in the same way as other hydroceles, namely, by injection, or radical cure by laying them open. Owing to their close connection with the epididymis, the cutting away of a portion of the sac is, in my judgment, far the best method to adopt.

Now we must consider the treatment of congenital hydrocele, which is found in boys and young children, where the fluid can be reduced into the abdominal cavity. When the child cries the fluid comes back again, very like a hernia. The extreme translucency will tell you that you have a hydrocele to deal with, and the sensation of reduction is very different from that of a hernia, "gurgling" being always absent. At the beginning I pointed out to you how risky it would be to inject these swellings, for the fluid might find its way into the peritoneal cavity, and set up grave inflammation. It is difficult for me to advise you regarding the treatment of congenital hydrocele. Great difference of opinion exists. Many surgeons recommend a truss, and state that the pressure

Digitized by Google

of the pad obliterates the opening. This is always a dubious and tedious method. I think it far better to perform an operation like that for radical cure of hernia, tying the neck of the sac high up, and sewing together the pillars of the ring. I make the incision longer and lower down, and at the same time excise the front wall of the sac.

Finally, I will make some remarks about complicated cases of hydrocele. First the combination of hydrocele with enlarged testis, so-called hydrosarcocele. This is often a very puzzling condition to students in a clinical examination. Hydrosarcocele is differentiated at once from hydrocele by the fact that when you lift the swelling up you find it extremely heavy, and when you begin to palpate it your fingers will displace fluid, and come down with a sudden impact upon the solid mass inside. The sensation is very similar to what is probably familiar to you in the medical wards in the case of dropsy with a cancer or cirrhosis of the liver. As you displace the ascitic fluid your finger will suddenly come upon the hard liver beneath. Hydrosarcocele may be non-translucent, except to a very limited degree in front. In the great majority of cases these sarcoceles are syphilitic, and are often bilateral. And the hydroceles may take very irregular form, being, indeed, mere collections of fluid limited by adhesions and false membrane. You may find hydrocele associated with tubercular disease of the testis. The hydrocele effusion is often very acute, and you may take this important clinical hint from me, that if a young man comes to you with hydrocele which he assures you is painful and has formed within a month or six weeks, it is almost always sure to be due to some disease of the testicle. Always tap such hydroceles, and carefully palpate the testicle before giving a complete opinion or prognosis. On several occasions, after tapping in such cases as I am alluding to, I have felt the peculiar nodular enlargement of the epididymis so significant of early tuberculous disease. In certain exceptional cases you get large collections of fluid surrounding malignant disease of the testicle, and the fluid may be bloody. Incision will clear the diagnosis up. Again, old men who use the catheter often get chronic epididymis and orchitis, and this condition may be associated with hydrocele.

In the treatment of hydrosarcoceles there is no objection to tapping if you do not wound the enlarged testis inside. The patient will be considerably relieved by the withdrawal of the fluid. It is not much good doing anything else until the testicle is radically treated. After drawing off the fluid with a fine trocar, if it is syphilitic, you strap the part with blue ointment and give iodide of potassium internally. In hydrosarcocele, when the testis is tuberculous, you remove at the same time the disease which causes it, and that cures the whole condition.

In old hæmatocele the consistence and shape of the swelling may be exactly like an ancient hydrocele. The patient may or may not tell you he has had a blow in that region; but it is a very important fact that many hæmatoceles are manufactured out of old hydroceles; that is to say that the gentleman who taps the hydrocele wounds the front of the testicle, or runs his trocar into a little vessel, and from that blood trickles slowly into the sac of the tunica vaginalis and gradually distends it. Thus what was formerly a hydrocele becomes converted into a hæmatocele. You will be quite safe in telling an examiner that it is impossible to diagnose between an old hæmatocele and an old hydrocele with thick walls except by an exploratory incision. When you incise it and you find thickened walls and altered blood and cholesterin, you cure the patient at the same time that you arrive at a diagnosis. I know that at examinations these cases give rise to trouble. The students cannot see through the swelling and become confused, and the refuge of exploratory incision is not enough present to their minds.

The history of sarcoma of the testis will be very different to that I have just related. Here there is a progressively increasing swelling, which has probably dated from six months to twelve months before, and it will be non-translucent. The story of a blow or "squeeze" is common enough. In addition, the veins over the scrotum are markedly enlarged, and there are frequently nodules of growth in the cord, and to the hand the swelling is very heavy, and as soon as incision is made into it the nature of the growth is determined.

That is practically all I have time to say to-day about hydroceles. My final word is for examination purposes. Remember that hydroceles are the common affections which produce scrotal swellings. You must not mistake them for solid tumours because they are hard and heavy. A hydrocele which is very much distended with fluid feels like a solid swelling. If you use your eyes, and notice the shape of the swelling, this always tells you what you have to deal with, and many of your difficulties of diagnosis will vanish.

Digitized by Google

SOME REMARKS* ON THE CAUSATION AND DIAGNOSIS OF ACUTE INTESTINAL OBSTRUCTION.

By J. H. BRYANT, M.D., F.R.C.P.

(*Concluded from p.* 122.)

3. The pathological conditions external to, but involving the gut, may be considered under four headings, viz:—(*a*) Strangulation by bands; (*b*) strangulation through orifices and internal herniæ; (*c*) kinking of the gut; (*d*) tumours pressing on the gut.

(a) Strangulation by bands.—This group is by far the most important, for it is one of the commonest causes of acute intestinal obstruction.

There are various structures which may act as bands, *e. g.* (*a*) peritoneal bands; (*b*) a Meckel's diverticulum; (*c*) the appendix vermiformis; (*d*) a Fallopian tube; (*e*) appendices epiploicæ; (*f*) the long pedicle of a tumour, *e. g.* ovarian, etc.; (*g*) omental cords; (*h*) an empty loop of intestine.

Peritonitis plays a most important *rôle* in the production of obstruction by bands for it may be the actual cause of the band or it may fix the free end of an already existing band or band-like structure, such as the appendix vermiformis, to some other part of the peritonial cavity, in such a manner that a coil of intestine becomes obstructed by it. Considered generally, there are several different ways in which obstruction may be brought about by bands:

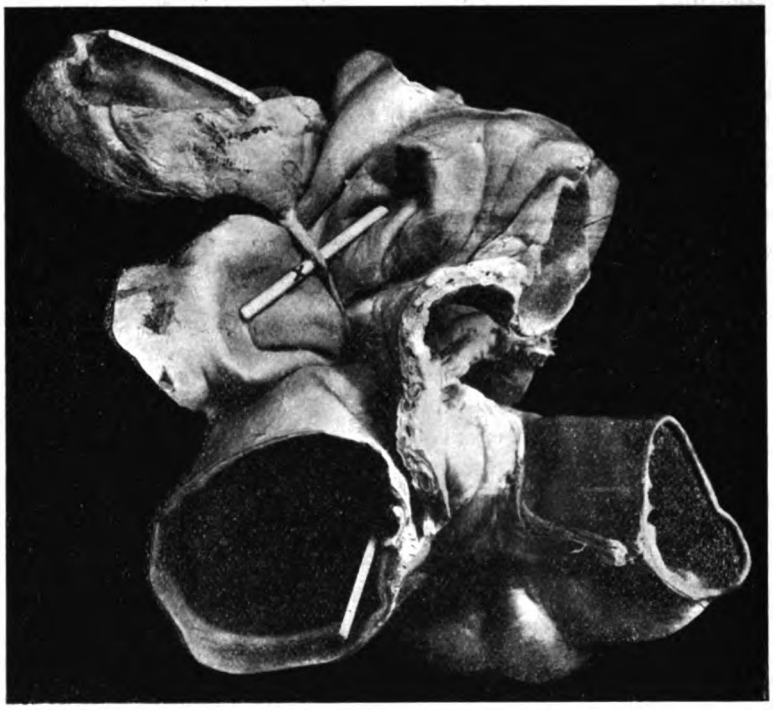


FIG. 7.—A peritoneal band indicated by a rod marked × placed beneath it.

1. If a band is short and unyielding, and it is firmly fixed between two points, a loop of intestine may become strangulated beneath it.

2. If it is short and fixed at both ends a coil of intestine may hang over it and become strangulated where it comes into actual contact with the band.

3. If the band is long, whether attached at both ends or free at one end, it may cause obstruction by forming a loop around a coil of intestine.

4. If the band is long and free at one end it may form a knot around a loop of intestine.

* Delivered before the Wimbledon Medical Society.

Digitized by Google

5. If one end is attached to a fixed structure, such as the brim of the pelvis, and at the other end to a piece of intestine, by contracting it may cause obstruction by pulling on and kinking the gut.

(*a*) *Peritoneal bands*, or false ligaments, are the result of previous attacks of peritonitis especially of localised peritonitis, such as appendicitis, pelvic peritonitis, tuberculous peritonitis, hernia, after abdominal operations, after injuries, etc.

between two coils of intestine, or between a coil of intestine and some fixed body, such as the brim of the pelvis or the uterus. Such a band may remain fixed at both ends, or one extremity may become so stretched and thinned that it gives way, and a cord attached at one end only results.

The length of these peritoneal bands may vary from about half an inch to twelve or fifteen inches. Treves mentions a case in which a band measured seventeen and a half inches. Here is a specimen

FIG. 8.—Meckel's diverticulum (D).

Coils of intestine may at first become adherent to each other or to other structures by means of plastic lymph: after a time normal or increased peristaltic movements may occur and lead to stretching of this plastic lymph. Many of the soft friable cords and bands produced in this way of course give way; but others, which are probably thicker and firmer, become still more elongated, and by means of the rolling movements of the intestines rounded. If organisation then follows, a firm cord-like band results, which may stretch

which shows a diverticulum attached by a band to the mesentery (Fig. 7) The band is a thin fibrous cord which is attached to the diverticulum an inch from its extremity on one side, and on the other side to the mesentery close to the origin of the diverticulum. Beneath the fibrous band a red rod has been passed, and it lies over a coil of ileum which is considerably constricted, although not actually strangulated. The patient was a woman aged forty-nine, who was admitted under the care of Mr. Howse for a femoral hernia with a history

Digitized by Google

of nine days' strangulation. Herniotomy was performed, but the patient died three days after admission. It was found at the post-mortem examination that the diverticulum of the ileum had formed the contents of the hernial sac. There was general peritonitis. Although in this case there was not actual strangulation and acute obstruction produced, it is an excellent specimen

FIG. 9.—Obstruction caused by an adherent appendix vermiformis.

to illustrate the manner in which acute intestinal obstruction may be produced by means of a peritoneal band.

(*b*) *Meckel's diverticulum* is a blind tube which comes off from the ileum from one to three feet above the ileo-cæcal valve; it is due to incomplete obliteration of, or to persistence of the vitelline duct. It may vary from a small almost imperceptible projection from the ileum, to a process nine or ten inches in length. It has the structure of normal intestine. It may cause obstruction in several ways: (1) If its free end is fixed, a loop of intestine may become strangulated beneath it; (2) if its free end is fixed, a loop may hang over it; (3) it may form a loop round a coil of intestine; (4) it may form a knot round a coil of intestine if it is long and its blind end is free and dilated: (5) if the blind end is attached to some fixed structure and there is much traction, it may cause kinking; (6) if it is short and becomes inverted it may lead to intussusception.

The specimen I have for you to see is of the lower part of the ileum (Fig. 8) "showing a diverticulum about four inches long, which is pervious and attached to the umbilicus. The diverticulum, which arises from the free body of the intestine, is bent over towards the mesentery, thus compressing and obstructing the bowel. Above the obstruction the gut is dilated, and below it is contracted."

The patient was a girl æt. 10, who was admitted

Digitized by Google

under the care of Dr. Wilks, for symptoms of intestinal obstruction of nine days' duration. Two days after admission she died. Acute peritonitis was found at the autopsy.

(*c*) *The appendix vermiformis.*—I shall illustrate obstruction produced by the appendix vermiformis by means of this specimen of the lower end of the ileum and cæcum (Fig. 9).

"The appendix is bent round over the ileum just above the valve, and its apex is adherent to the free border of the bowel two feet higher up. At these two points the gut is thus tightly constricted by a band, and the intervening coil of ileum was in the recent state deeply congested."

The patient was a man æt. 46, who was admitted, under the care of Dr. Taylor, for abdominal pain and vomiting of ten days' duration, which came on quite suddenly. Death took place three days after admission. In addition to the above-described condition, tuberculous consolidation of the lung and aortic incompetence were found.

(*d*) *Fallopian tube.*—If the free end of a Fallopian tube becomes attached to some other structure, a loop may be formed under which a coil of intestine may become obstructed. This specimen will illustrate the condition (Fig. 10). It consists of "the female pelvic viscera with a portion of the ileum mounted to show the right Fallopian tube turned upwards and backwards, and firmly attached by its fimbriated extremity to the rectum. A loop is thus formed, through which a coil of intestine about six inches in length has passed and become strangulated. Douglas's pouch is obliterated by old adhesion."

The patient was a woman æt. 60, who was admitted under Dr. Goodhart with intestinal obstruction. The bowels had been confined for three weeks before admission, and for the seven days previous to her coming in vomiting had been well marked. She was operated on two days after admission, and died on the following day.

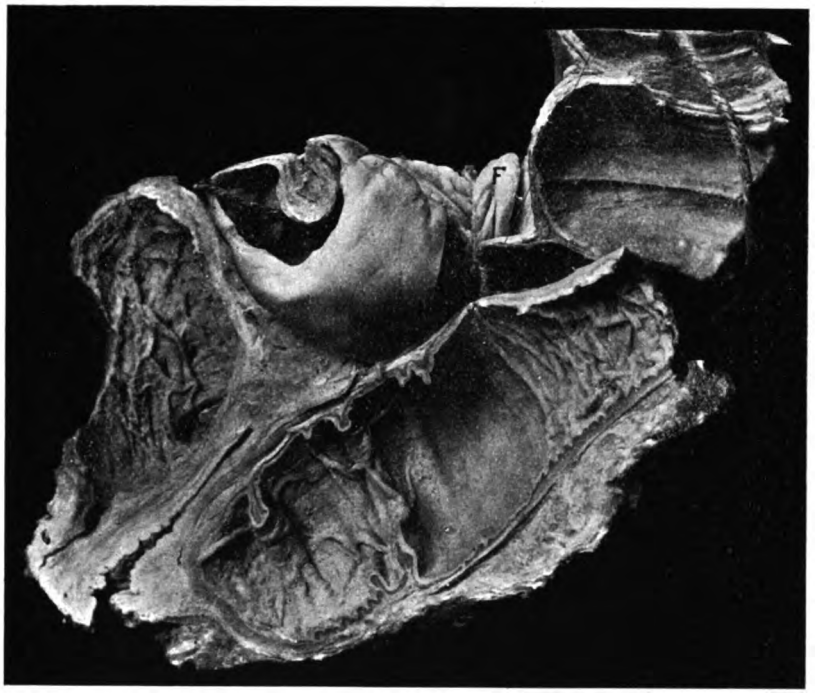


FIG. 10.—Obstruction caused by an adherent Fallopian tube (F).

The strangulated portion of gut was the ileum just above the cæcum.

(*e*) *Appendices epiploicæ.*—The ends of two of the appendices epiploicæ may become adherent, and through the loop thus formed a coil of small intestine may slip and become obstructed, or possibly the appendices may rest one on each

Digitized by Google

side of a loop of intestine and become adherent, and contract and produce obstruction in this manner. Here is a good specimen of obstruction thus caused (Fig. 11). It consists of a portion of the sigmoid flexure of the colon coated with fat, and presenting large appendices epiploicæ. Two of these are adherent to each other by their tips,

FIG. 11.—Obstruction caused by adherent appendices epiploicæ (A).

and through the aperture thus formed a loop of ileum five inches in length has slipped and become strangulated.

The patient was a woman æt. 48, who was admitted under Dr. Perry for intestinal obstruction of six days' duration. She was operated on, but the cause of obstruction was not discovered. She died on the following day. The piece of gut involved was found to be the ileum four inches above the cæcum. With regard to this specimen, I am inclined to think that a piece of the ileum must have first of all been surrounded by two appendices epiploicæ, and that afterwards their tips became adherent, and then more small intestine slipped through until strangulation occurred. It seems difficult to understand how the loop of ileum could have slipped through such a small movable ring as I have shown you.

(*f*) *The long pedicle of a tumour.*—Ovarian tumours, uterine fibroids, and floating kidneys often have long pedicles, which may act in much the same way as the bands I have already alluded to, and give rise to acute intestinal obstruction. For instance, a long pedicle may form a loop around a portion of intestine, or, if the tumour becomes fixed, a loop of intestine may become obstructed beneath the pedicle, or may hang over it, and thus become obstructed. I have not personally come across a case of obstruction from this cause, but there are several instances on record.

(*g*) *Omental cords* may be formed if a portion of the free edge of the great omentum becomes firmly attached to some distant part, such as the brim of the pelvis, the uterus, or a hernial sac or orifice. As a result of this fixation a certain amount of constant traction is exerted on the lower border of the omentum, which becomes, in consequence, stretched, narrowed, and rounded into a band. Bands formed in this way are usually large and thick. Obstruction is produced in the same manner as with other forms of bands.

There is another interesting way in which a band may be produced. A small portion of intestine, as a result of peritonitis, may become attached to a solid or fixed structure, in two places close together, and as a result a small aperture may be formed bounded partly by the solid structure, and partly by the portion of intestine between its two points of adhesion; a loop of intestine may slip through an aperture thus formed, and become strangulated. I hand round to you an extraordinary, and, I believe, unique specimen of this kind (Fig. 12). It consists of a portion of the ileum, the cæcum, the appendix vermiformis, and a lithopedion. For two feet immediately above the ileocæcal valve the ileum is completely collapsed, this portion being strangulated as a result of having

Digitized by Google

passed through a small aperture formed by a short piece of the ileum immediately above having become adherent to the body of the lithopedion in two places.

The patient was a woman æt. 40, who was admitted under the care of Dr. Perry on November 28th, 1899, for intestinal obstruction. She had been constipated, and had suffered from abdominal pain for ten days previous to her admission. Two and a half years before there was a history of five

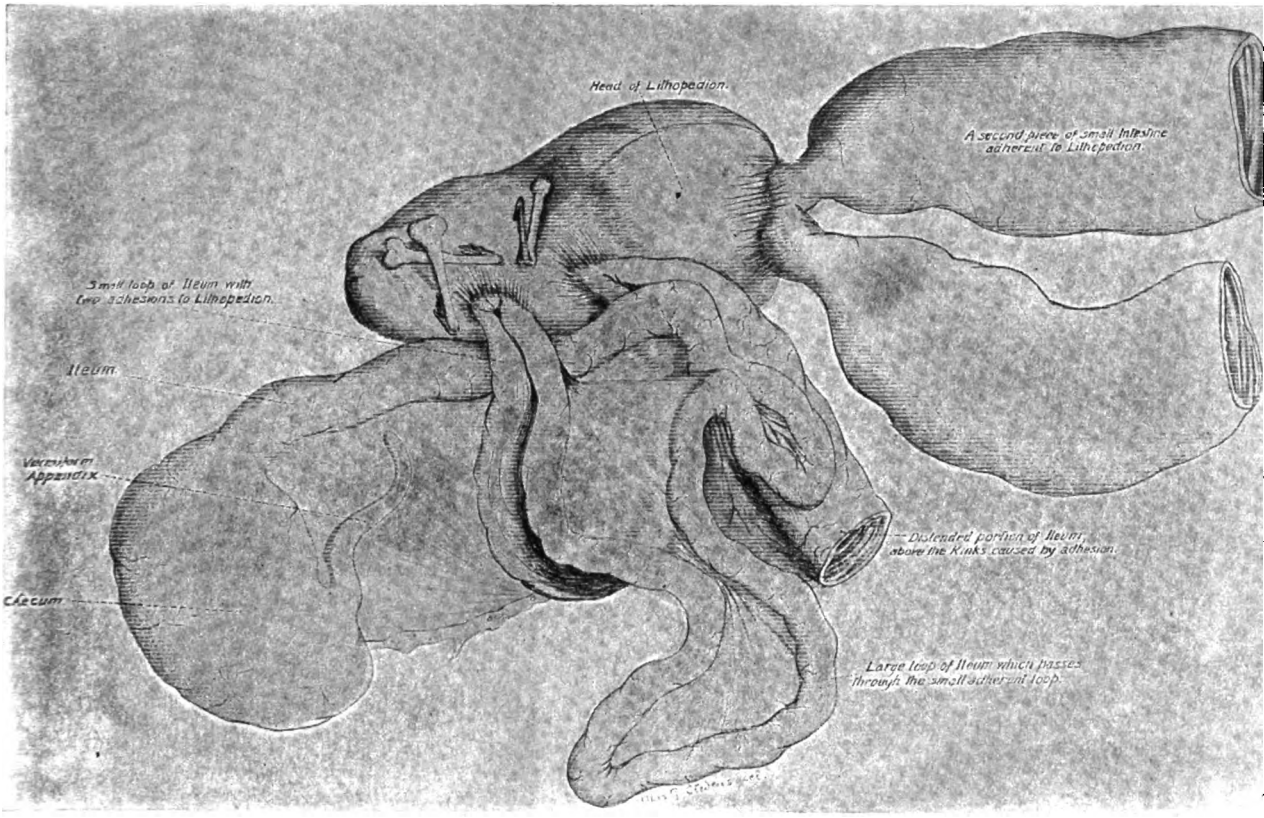

Head of Lithopedion.
A second piece of small Intestine adherent to Lithopedion.
Small loop of Ileum with two adhesions to Lithopedion.
Ileum.
Vermiform Appendix.
Distended portion of Ileum above the Kinks caused by adhesion.
Cæcum.
Large loop of Ileum which passes through the small adherent loop.

FIG. 12.

months amenorrhœa. As far as she knew she had never been pregnant. On the 29th she was operated on. A hard mass (the lithopedion) was felt, and considered to be a growth binding down and kinking the intestine. She died early the next morning.

(b) Internal herniæ.—Strangulation and obstruction may be caused by portions of small intestine slipping through holes or slits in several of the peritoneal structures, *e. g.* in the mesentery, omentum, broad ligament, suspensory ligament of the liver, etc. The causation of these holes and slits is very obscure; some may be the result of injuries. I have already shown you a specimen which might, perhaps, have been considered under this heading, of a portion of the ileum which was supposed to have slipped through the aperture formed by two of the appendices epiploicæ having become adherent to each other at their free extremities. Mr. Treves mentions an extraordinary case of Mr. Holmes, in which a loop of the lower part of the ileum was strangulated through a hole in what appeared to be an appendix epiploica. I have here a piece of omentum showing a small aperture about an inch in diameter, through which a loop of ileum had slipped and become strangulated.

The patient was a man æt. 61, who was admitted under the care of Dr. Moxon for symptoms of acute intestinal obstruction of three days' duration. He died three days after admission, and at the post-mortem examination it was found that the

Digitized by Google

last three feet of the ileum had become strangulated as a result of having slipped through the aperture in the omentum.

In addition to the above there are the various forms of internal hernia, which I shall only briefly describe, using Treves's classification: (*a*) Diaphragmatic; (*b*) hernia into the fossa duodeno-jejunalis; (*c*) hernia into the foramen of Winslow; (*d*) intersigmoid hernia; (*e*) perityphilitic hernia.

In *diaphragmatic hernia* the opening in the diaphragm may be congenital or may be due to injury. The organs which are most usually herniated are the stomach, transverse colon, and then the small intestine, liver and spleen.

The distinctive signs of this condition are falling in of the abdomen, dyspnœa, displacement of the heart, and distension of one side of the chest with hyper-resonance and suppression of the respiratory sounds.

Hernia into the fossa duodeno-jejunalis, or as it is more commonly called, retro-peritoneal hernia. The first case of this kind was recorded by Treitz in the 'Guy's Hospital Reports' for 1872. The fossa is formed by a fold of peritoneum at the junction of the duodenum with the jejenum. The whole of the small intestine may be implicated.

Hernia into the foramen of Winslow.—The small intestine slips through the foramen of Winslow into the lower peritonial cavity behind the stomach.

Intersigmoid hernia.—A funnel-shaped opening called the intersigmoid fossa may be formed by the meso-colon over the bifurcation of the left iliac vessels. The symptoms may be acute and rapidly fatal.

Perityphlitic hernia.—Pouches may be found by the peritoneum, in the region of the cæcum. A few cases (about 12) only are on record. The symptoms are usually acute.

(o) **Kinking of the gut.**—I have already mentioned that if the free end of a diverticulum is attached to some fixed point, such as the umbilicus, the intestine at the point where the diverticulum arises may be obstructed by acute kinking, the result of traction. A diverticulum may be responsible for acute kinking in another way. It may become much distended with intestinal contents, and give rise to such acute bending of the ileum as to completely block its lumen.

Kinking is more frequently produced by peritoneal adhesions. The adhesions are usually short and firm, and fix the portion of the intestine involved to the abdominal parietes, pelvis or pelvic viscera. Pelvic peritonitis, hernia, or appendicitis are the commonest causes of it.

This specimen (Fig. 13) of a coil of ileum adherent to the abdominal wall near the umbilicus,

FIG. 13.—Kinking from peritoneal adhesions.

shows well how kinking of the gut may be produced. At the point of attachment the ileum forms a V-shaped bend, "and the portion of bowel which forms one limit of the V has become laterally adherent to the fold of the mesentery stretched between it and the other limb."

The patient was a man æt. 43, who was admitted under the care of Dr. Bright in 1828 for intestinal

Digitized by Google

obstruction of two weeks' duration. He died three days after admission.

At the autopsy the cause of obstruction was thought to be the kinking of the ileum.

There is another condition which may cause kinking, and that is contraction of the mesentery. Here is a specimen of kinking from this cause (Fig. 14).

FIG. 14.—Kinking (K) from contraction of the mesentery.

It consists of "a portion of jejunum with its mesentery, the lymphatic glands of which are enlarged. The bowel is seen to be sharply bent on itself at a spot where the mesentery is shortened by old inflammation. Above the obstruction thus produced the intestine is considerably dilated."

The patient was a boy æt. 10, who was admitted under the care of Dr. Hale White for acute intestinal obstruction. He died on the fifth day after the onset of the symptoms.

With reference to the diagnosis of strangulation by bands, the onset is always sudden, and in about 70 per cent. of the cases there is a previous history or evidence of peritonitis. Pain is one of the earliest symptoms, and is usually severe, and in many of the cases is most intense in the umbilical region. Distension may or may not be present, it depends a good deal on the part strangulated; the higher up the strangulation the less the distension. Vomiting is another symptom which occurs early, is usually severe, and soon becomes stercoraceous. Collapse and prostration soon appear. Constipation is constant and absolute. Tenesmus is absent.

The various forms of internal herniæ and acute kinking are almost impossible to distinguish from obstruction and strangulation by bands.

(d) Pressure of tumours.—A tumour may give rise to acute obstruction by pressure on a portion of the intestine if a sudden enlargement arises in the tumour, *e. g.* from hæmorrhage into its substance, or if, in the case of an ovarian with a pedicle, the pedicle becomes twisted, and the tumour becomes enormously congested as a result, or if a tumour suddenly becomes jammed and fixed into the pelvis. It is a very rare cause of acute intestinal obstruction. In the 'Medical Review' for October, 1900, there is an abstract of a paper by Schalika, based on two cases of this nature.

The tumour in the first case appeared to be a hæmatoma connected with the right broad ligament, but turned out to be a multilocular dermoid, containing a number of blood-clots in addition to fat, bones, hair, and colloid material.

The tumour in the second case was an ovarian with a twisted pedicle. The obstruction was relieved by the removal of the tumour.

The diagnosis can only be surmised if the presence of the tumour is known.

I had intended to enter more fully into the question of diagnosis, but I see that I have already very considerably exceeded my time.

(The photographs are from specimens in the Guy's Hospital Museum, and the descriptions are based on the accounts given in the museum catalogue, edited by Drs. Perry and Shaw.)

THE cumulative action of digitalis is due to its contracting the arterioles, thus shutting off nutrition. The drug is therefore both a useful and a dangerous remedy, and one that has a very limited range of usefulness. It is only of use in lesions of the mitral valve, and even then only for a short time. It should only be used when there is low arterial tension and marked venous engorgement, and as soon as these conditions are overcome its action should be suspended.—Dr. PORTER, *American Medicine*, April 27th, 1901.

Digitized by Google

DIAGNOSIS IN WRY-NECK.

MR. C. B. KEETLEY.

According to Mr. C. B. Keetley in his recently published work on "Orthopædic Surgery," the most important diagnosis by far, and one sometimes difficult in wry-neck is the distinction between true wry-neck and cervical caries.

In true wry-neck mobility is limited in range, but perfectly free within the limits. There is no spinal deformity, except *lateral* curvation. But the rotation which accompanies this may in the upper part of the cervical spine cause a postero-lateral prominence, which might be mistaken for the results of subluxation or inflammatory thickening. The position of the head and neck shows the characteristic inclination to one side, combined with a twist to the other. There is no pain caused by pressing the head firmly or jarring it moderately, consequently the patient usually looks fearless, and perhaps bright and merry. He picks up an article from the floor with as much activity as a healthy child. There are no abscesses or sinuses, except that in rare cases they may be accidentally present.

In cervical caries there is as a rule, little or no *free* mobility at all; on the contrary, stiffness. Frequently a prominence can be felt (if not seen), posteriorly, of the carious vertebræ. It may be difficult to feel anything abnormal if the atlas or axis be the bones affected, but then the stiffness, and perhaps tenderness, of the atlo-axoid and atlo-occipital joints will be marked. It is rare for the position of true wry-neck to be accurately simulated. There is much more frequently a mere bowing forward of the head, bringing the chin toward the sternum. Pain *is* caused by jarring the head or firm pressure on it. Consequently the patient is cautious and timid, both in moving it himself and permitting it to be interfered with by others. He may support his chin in his hand. He lowers himself cautiously, with a view to avoid jarring the spine or throwing his head forward. Frequently sinuses or other signs of suppuration, past or present, are in evidence.

Want of space alone prevents our printing an extended notice of Mr. Keetley's scholarly volume entitled "Orthopædic Surgery," published by Messrs. Smith, Elder and Co., but the valuable nature of Mr. Keetley's teaching can be gauged by the perusal of the foregoing paragraphs from his work.

ERYTHROMELALGIA AND ARSENIC.

DR. T. N. KELYNACK and MR. W. KIRKBY.

Erythromelalgia is an affection characterised by two main features: erythema and pain. It has been very well marked in a very large number of the cases described in the book just issued by Dr. T. N. Kelynack and Mr. W. Kirkby on "Arsenical Poisoning in Beer Drinkers."

The erythema has varied much in intensity. In some it has been slight and easily overlooked, while in others it has been rather that of an acute dermatitis with, in some instances, a certain amount of œdema. The colour has varied from a bright red to a purple. Most usually it has been of a purplish red. The distribution is very characteristic. It is almost always more or less symmetrical. It involves the soles and borders of the feet most markedly. In some the hands present a somewhat similar condition. The erythematous border, often slightly undulating and frequently clearly defined along the edges of the feet, has generally been readily recognised in this class of case. The dorsum of the foot is usually not involved. The erythematous areas have often been found covered with sweat, and frequently have felt cold both to the patient and to the examiner. In the acute stages they have generally been described as "hot and burning." The pain in many instances has been severe; movement of any kind has always aggravated it, the erect position has usually increased it, and walking in severe cases has been rendered impossible. In many instances patients have not been able even to bear the weight of the bed-clothes on the affected feet. In the case of a child a little over two years of age, where they found distinct erythema of the feet, the first thing to attract attention to the condition was the fact that the little sufferer was always wanting to take her shoes and socks off. Almost every degree of erythromelalgia has been met with. In almost all cases pressure has increased the pain, and in acute cases even merely touching the foot caused the patient to exclaim. Sometimes exposure to cold has brought temporary benefit.

The book "Arsenical Poisoning in Beer Drinkers," published by Messrs. Balliere, Tindall, and Cox, is an admirable example of what a work of this sort should be, and the remarks quoted above indicate sufficiently the excellent manner in which the authors have acquitted themselves of their task.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 452. WEDNESDAY, JUNE 26, 1901. Vol. XVIII. No. 10.

CONTENTS.

PAGE

*With Mr. Stanley Boyd at the Hospital for Consumption and Diseases of the Chest, Brompton 145

*Present Day Skiagraphy. WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL. Demonstration I 152

*Four Clinical Lectures on some Affections of the Kidney. By A. H. TUBBY, M.S., F.R.C.S. Lecture III 157

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum: Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

WITH MR. STANLEY BOYD AT THE HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON.

MARCH 13th, 1901.

LADIES AND GENTLEMEN,—In giving a lecture on "cases in the wards" one has to depend on the cases which happen to be there. I have nothing very startling to lay before you, but I think I have some cases of interest. Those which I want to bring before you first illustrate inoculation of the skin with the *Bacillus tuberculosis*. In many cases we have to theorise as to how infection takes place. For example, you find that there are bacilli in the bronchial glands; you search through the lungs very carefully, and through the mucous membrane of the respiratory passages draining to the bronchial glands, and you may find no lesion. How, then, did the bacilli get to these glands? You infer that they passed through the mucous membrane of the pulmonary tract somewhere or other, leaving no primary lesion at their point of entry, or if there was any little disturbance during their passage, that it completely subsided, and left no post-mortem evidence. But in the following cases there is no need to theorise at all; the inoculation of the *Bacillus tuberculosis* at certain points in man is clear, and in one of the cases we can follow the course of the disease most admirably. The first case I cannot show you, but beyond a normal scar there is really nothing now to show. The case was that of one of our nurses here, who, in March or April, 1900, was carrying a tray of used spitoons; she fell, and a broken spitoon made a clean cut on her left wrist, in front of the radial artery. It healed readily, but not soundly. In about a fortnight the dressing was taken off, and a scab was left. When this was removed a little central hole was found in the scar, and from and around this, redness and infiltration of the skin slowly spread; the soreness then became more marked, and the central

Digitized by Google

hole remained until August. At that date I was asked to see her. I found that she had an area of thickened skin the size of a fourpenny piece, red, dry, and somewhat warty on the surface, with a well-defined edge. I do not remember seeing the little hole which she describes in her history. The glands were not affected, and there was no evidence of spread of the infection to the lymphatics of the forearm; the whole case would be summed up by saying that she had an inflammatory thickening of the skin at this spot. I cut it out completely, and brought the edges together. The little wound healed by first intention, and she is perfectly well and has had no trouble since. You will be able to look at a microscopic section afterwards, and you will see that the disease was the result of infection by the tubercle bacillus.

The next case was that of a lady whose age was about thirty-five; she had been suffering for many years from chronic phthisis in the right lung. In January, 1897, fifteen months before I saw her, during an exacerbation of her phthisis—with much expectoration, which she always received upon a handkerchief—she caused a slight abrasion with a lump of sugar on the extensor aspect of the first phalangeal joint of the right index finger. That abrasion never healed; and, although no microscopic examination was made of any of the parts removed, the clinical course renders it practically certain that this was because tubercle bacilli were inoculated from the handkerchief upon the sore. For some months no attention was paid to the sore; finally she saw a surgeon, who rang the changes on cod-liver oil and iron, and iron and cod-liver oil. That went on for a considerable time—I think about a year—and no improvement took place; on the contrary, the disease was spreading in a most characteristic way. The first thing noticed was, that some few months after the infection, a considerable swelling formed somewhat acutely in the situation of the internal supracondylar glands. Probably there was a mixed infection of the glands, septic or pyogenic, as well as tubercular. No suppuration occurred, however, and after a time these swellings subsided, though not completely. Next she noticed an elongated patch of infiltration just below the elbow, over which the skin was adherent and reddened around the radial lymphatics. Next a similar patch formed on the dorsum of the hand, over the second interosseous space, which ulcerated slightly. The last result the patient noticed was a small mobile lentil-like subcutaneous nodule on the ulnar side of the olecranon process; it seemed obviously to be in some lymphatic vessel. This was the condition of things when I saw her in March, 1898; January, 1897, having been the date of infection from the handkerchief. I then found in addition two or three enlarged lymphatics behind the pectoral muscle. So this case showed the way in which tubercle spreads by the lymph path from an evident primary lesion to the first and second group of glands above it; and, what is uncommon in the skin and subcutaneous tissue, there were here and there in the course of the lymphatics small foci of tubercular lymphangitis due to arrest and growth at these spots of bacilli. It is interesting to note that they did not appear until after the lymphadenitis, which probably slowed the circulation in some lymphatic vessels.

Under ether I excised the edges and scraped the base of the primary lesion, then applied pure phenol, and dressed it with iodoform. I excised all affected glands, patches, and nodules. Everything united by first intention except the finger; that did not heal, in spite of frequent and free applications of pure carbolic and iodoform. Ultimately I excised it completely. I did not do so at first because I feared the effect on the finger of such a free removal of skin. It now healed slowly but soundly—some sixteen months after the first operation. During most of this time fresh little shot-like nodules kept appearing along the course of the radial lymphatics. One by one they were excised under cocaine, seven or eight such operations being done. Once or twice I had to repeat an operation for recurrence *in loco*, possibly because the first excision was incomplete; for after the production of cocaine œdema all evidence of the little nodule, to sight or touch, was lost. The patient, fortunately, was plucky and determined, for the course of treatment was long and especially trying to one in feeble health. She has now been free from recurrence for more than eighteen months, although she has had pleurisy and some advance of her chest trouble.

The lessons to be learnt from these cases are: (1) that timely excision of tubercular sores is the proper treatment, and (2) that with patience and perseverance we may succeed in arresting the

Digitized by Google

disease even where it has got a long start. It is, however, only fair to point out that in the second case the patient, though liable to tubercle, was evidently resistant in a fairly high degree.

I pass next to a case of direct infection of a tendon-sheath with tubercle bacilli in the person of the post-mortem porter (æt. 62) in this Hospital, whose duty it is to stain the sputum for examination. After employing a steel pen to spread some sputum, known to be virulent, upon a cover-glass, he left it projecting beyond the edge of the table, and brought his hand forcibly against its point. The nib was driven into the proximal end of the flexor sheath of the ring finger. This occurred in the middle of June, 1900. He immediately tried to run fuming nitric acid into the wound caused by the pen, and no doubt he disinfected the surface by that means, but not the depths. A hard lump gradually formed in the palm and extended down along the ring finger, which became flexed, stiff, and painful. He was treated by means of a palmar splint, and was sent to Brighton for some weeks. He got no better. There was an interregnum at the Hospital, and I did not see the man until August. Then the finger was contracted, and the middle and little fingers also were coming down into the palm. A long, low, tense swelling occupied the whole length of the flexor sheath of the ring finger. The diagnosis was obvious; it was a case of tubercular teno-synovitis. The treatment was equally clear. The man had had the advantage of the best hygienic conditions which could be provided for him; he had been to the seaside with nothing to do except to look after himself and get well. But he got steadily worse. The next stage would probably have been that the tubercular tissue would have burst through the ring-finger sheath, infecting other palmar structures, especially the common sheath of the flexors at the wrist; and there was no knowing where the trouble would cease. The complete removal of the disease was urgently necessary. I therefore split the tendon sheath almost completely open, and came down upon a quantity of granulation tissue which showed no cheesy degeneration. It was not vascular, and you know tubercular granulation tissue is not so as a rule. I cleaned this out as perfectly as I could with a knife and forceps, raising the tendon and having it kept tense while I cleaned it all round. Then I applied pure carbolic to the whole surface of tendon and sheath. You will appreciate that it was of far less importance to retain the movement of the finger than it was to obtain the cure of the disease. The result was absolutely satisfactory, as you will see. It is now more than six months since the operation. Extension of the finger is somewhat limited by the long longitudinal scar on its palmar aspect. But he has fair and very useful movement, and he is perfectly free from recurrence. You know that the prognosis of tubercle gets worse as age increases, and therefore this result may be considered very satisfactory. With regard to painting infected tissues with pure phenol, it certainly adds to the probability that all is destroyed; the amount of irritation which it causes is by no means great, and very soon subsides. When the wound is aseptic it causes little or no trouble in the healing.

A section of the granulation tissue removed will show you "giant-cell systems" in large numbers and great perfection.

These cases of direct infection are very interesting, but they are by no means common. Previously in my experience I should have been able to demonstrate it only in the case of the lady I have mentioned to you. But directly I came here, you see, I met with two cases. The resident medical officer tells me that during his time here, he had not previously seen a case. In Koch's first paper on the *Bacillus tuberculosis*, when he was gathering together evidences of infection, he quoted a case very similar to that of the nurse I first mentioned, only it went on as far as the second case I told you about. This woman was a cook to a gentleman suffering from phthisis, and it was part of her duty to look after his spitoon. She cut her thumb accidentally on a broken spitoon and got a sore there, whence lesions spread all up her arm to the axilla. All were excised, as in my patient, and with an equally satisfactory result. But in one important respect Koch's case was the more complete, for the tubercular bacillus was sought for and found in all the lesions.

I want to pass now from teno-synovitis of one of the flexor tendon sheaths of the hand to the common "compound ganglion" which we see most commonly round the flexor tendons in front of the wrist. Another common situation is round

Digitized by Google

the extensors on the dorsal aspect. We have a case of each variety here. The first case is that of a man æt. 30, single, a French polisher by trade. He has had cough for the last two years and two attacks of hæmoptysis, with infiltration of the right upper lobe, and probably of the lower. Six months ago some of his left fingers swelled, and, later, the front of the left wrist. The swelling in the fingers disappeared, showing that it is possible for such an affection to subside of itself; for, I take it, the swelling of the fingers was due to teno-synovitis of the flexor sheaths. The right hand went through the same course; swelling, first of some of the fingers, and then of the great sac in front of the wrist. On both sides the swelling remains in front of the wrist. The result of the swelling was first of all a certain amount of pain, which may be very considerable; I have known it sufficiently severe to prevent sleep. Another effect is limitation of movement, chiefly of the fingers whose tendons are most surrounded by the diseased tissue; for it does not follow that because this sac is affected that the whole of it is involved uniformly; indeed, the distribution of the synovial membrane in front of the wrist seems to be extremely variable in different persons. Operations bring to light conditions which ordinary anatomical teaching does not explain. We sometimes get wasting of the muscles of the thenar and hypothenar eminences, and it is difficult to account for this. In one case I operated upon I found a considerable, inch-long, oval swelling of the nerve, presumably due to infiltration of the nerve—to a localised tubercular neuritis; and in that case the thenar eminence was greatly wasted. In other cases the hypothenar eminence is wasted, and the hand assumes the interosseous position, as if the deep ulnar nerve were affected. But it is difficult to see how that can be. In the patient I am showing you there was loss of power of flexing the metacarpo-phalangeal joints, that being a function of the interosseous muscles. But he had not lost the power of extension of the interphalangeal joints, which also is effected by the interossei. He had the usual swelling above the annular ligament well marked: very little or no swelling in the palm. The movements of the fingers were rather more limited on the right than upon the left side, and both hands were practically useless for his trade. I treated this man by splitting the sheath open nearly to the bottom of the annular ligament, choosing the right side to begin with. Each tendon was carefully cleaned—those of the little finger and ring finger being most affected; but all showed a coating of swollen synovial membrane, even the long flexor of the thumb. The median nerve, too, was distinctly wrapped in thin tissue, and I had to split the annular ligament almost to its lower border to clean the median nerve satisfactorily. I saw the branch to the thumb muscles being given off, and fear that in some way I must have injured the nerve, for since the operation the abductor and opponens pollicis have wasted more markedly, and sensation in the thumb and forefinger is still impaired. Iodoform was freely applied; the annular ligament, deep fascia, and skin were closely sutured, and the wound was sealed with collodion. Healing took place by first intention, and the man was encouraged from the first to perform all the movements of the fingers and thumb, and we are waiting until this hand should become sufficiently useful to enable him to dress himself before operating on the other hand. You can now see what amount of movement he has seven weeks after operation. He has now complete extension of the fingers, which he had not at the start; flexion is still very imperfect, but he can flex the metacarpo-phalangeal joints somewhat, as well as the inter-phalangeal, and this movement is improving. Even if no further improvement occurred, there is more extensive movement now than there was before the operation. We can insist upon getting extension by passive movement, but not on his making flexion. Flexion must be made by his own voluntary effort; we have no means of flexing for him. Hitherto I have kept the fingers midway between flexion and extension, and have taken care to have them extended. In future, I think, I shall allow the fingers to rest in a position approaching full flexion, seeing, of course, that they are fully extended passively twice or thrice daily. I began treating compound ganglia by excision in 1890, and I think that the method is likely to rid the patient of the disease, to preserve all the movement present at the time of operation, and often to gain a little.

I have another case, a dorsal one, with characteristic swelling around the extensor tendons, and no sign of disease of the wrist. He has bad phthisis, and is in very poor condition. Not

Digitized by Google

feeling inclined to do any cutting operation upon his hand, I am endeavouring to treat the ganglion by weekly injections of a sterile 10 per cent. solution of iodoform in almond oil, which I here show you. The difficulty, of course, is to get it absolutely sterile. The dispenser here raises the almond oil to a considerable temperature, and at the same time sterilises the bottle to contain it. To the cooled oil is added the proper quantity of sterile iodoform. To obtain this, heat cannot be used as it sets iodine free. It is best cleaned by prolonged soaking in 1 in 20 carbolic lotion. The injection is carried out by means of a beautifully made French syringe, entirely of glass, which can be boiled without injury. The needle is a fair-sized one. The quantity injected is 2—4 c.c. of the 10 per cent. solution of iodoform. I will give this patient an injection after the lecture. You will note that it causes some pain, apparently by tension; but it causes much less than an ethereal solution.

The next cases are examples of perichondritis and chondsitis of rib cartilage. I have no patient here to show the condition before operation, but I have two men here in whom a diseased cartilage has been excised. One of them is not quite healed, but the other is.

The first case was that of a man aged forty-two, suffering from chronic phthisis affecting the right upper lobe and the whole of the left lung. During three or four weeks previous to my seeing him, an inflammatory swelling had been forming about the left sterno-clavicular articulation. The cause was unknown. When seen there was a red swollen area as large as the palm around and below the left sterno-clavicular joint, firm and indurated marginally, "pointing" over the first sterno-chondral articulation. The sterno-clavicular joint was not affected. It therefore seemed probable that the primary cause of this patient's trouble was some inflammation of bone or cartilage about the inner end of the first rib. I raised a flap of skin and pectoral muscle, and, deep to it, entered a large focus of cheesy pus. At the bottom of this, surrounded by swollen tissues and at a great depth from the surface, I found the first rib and rib cartilage bare for over an inch. Working outwards towards the subclavian vein, I excised about an inch of the bone, and would have taken away more if I could have found any instrument to my hand which would have enabled me to overcome the mechanical difficulty which the case here presented. Then I worked inwards, and removed all the cartilage which seemed to be involved in the abscess wall. In doing this I found, to my surprise, behind the cartilage quite a considerable collection of cheesy pus, which was pressing back towards the pleura. The cavity would have held a hen's egg, and ran downwards and inwards behind the sternum. It was carefully cleaned out, pure carbolic was applied to its surface, the cavity was plugged with iodoform gauze, and the flap fixed in position. Everything went on satisfactorily at first, but later it became obvious that a piece of cartilage towards the sternum was remaining bare. Therefore, on November 12th, I had him again placed under an anæsthetic, and removed the rest of the first cartilage and even a small portion of the site of the sternum where the cartilage was attached. The deep cavity seemed quite to have filled up. The wound was dressed as before. On November 20th, when he went home, there was a small sinus leading to bare sternum. Now you see the wound is healed, apparently soundly. Unfortunately no examination of the pus or tissues removed was made in this case; but it was probably a case of tubercular periostitis and perichondritis.

Here is another man, a labourer, æt. 36. He gives no family or personal history of phthisis; he does not seem to have suffered from typhoid or syphilis. He attributes his trouble to a fall, eight months before I saw him, which drove a tobacco-box in his pocket against his chest. Shortly after the blow a swelling formed, and in about six weeks he had it opened. Pus was evacuated, but it did not heal. After a good many weeks, during which the cavity was plugged, he was anæsthetised and erasion was practised. It then healed up and remained healed for six weeks. Then it burst open again, and after a few weeks more he came under my care. He had two small sinuses leading to the left sixth cartilage; the tissues around were much matted. Raising a small flap, I found that a probe passed from sinus to sinus, right through the sixth cartilage horizontally. This was a very singular cartilage. It broke in removal, but I have had it pieced together to show you. Externally you see that I cut through perfectly normal rib cartilage; but the inner two inches are much

Digitized by Google

enlarged, and converted into an irregular, rugged mass of bone, which quite filled the interval between the fifth and seventh cartilages near the sternum. In the centre is a considerable cavity, full of granulation tissue, round which the bone forms a rather thick shell. In the shell were two openings opposite each other, each being in connection with one of the sinuses opening in the skin surface. In short, the specimen, as I read it, shows that the disease was primarily central in the cartilage. Central disease, of course, means the presence of a vessel penetrating to the centre, and embolism of some micro-organisms which start the disease into the vessel. Of course you know that cartilage for the most part is non-vascular; but rib cartilage is so. These thick rounded masses of cartilage have a few vessels penetrating them, and therefore it is possible to get a central disease of a rib cartilage. The specimen shows further that "expansion" may occur, as it does in bone; *i. e.* new tissue is laid down peripherally by the perichondrium, while the granulation tissue causes central absorption. In this instance the new tissue on the surface ossified, but it does not always do so. Early in 1899 I removed the sixth right costal cartilage of a woman, aged forty. Six months earlier she had had a "cyst" excised, and a sinus had persisted since, which led to cartilage. In this case the sixth cartilage was "expanded" in its middle one and a half inch; the central cavity was large, the wall thin and cartilaginous, and perforated at two or three spots. There was no ossification here. The woman attributed the abscess to the pressure of the handle of an invalid chair.

In the first of these cases I had sections cut and stained for tubercle bacilli; none were found, though some very thick bacilli and many cocci were present. The structure of the exudation lent no support to the idea that the inflammation was tubercular.

I can add another case of disease of rib cartilage in which the typhoid bacillus was regarded as (but was not proved to be) the cause. At the end of fourteen weeks of typhoid fever and pneumonia, while still in hospital, a woman of thirty-eight developed an abscess below and internal to her right breast. It was opened but did not heal. Three months later she came under my care. I cut down and removed her fifth cartilage, and in it I found a considerable sharp-edged and sharply bounded cavity, the appearance of which suggested that it had started around a penetrating vessel. I have not met with a case of disease of rib cartilage which seemed to be caused by syphilis; but as there is syphilitic disease of sternum and rib, it is possible that syphilis may cause disease of the cartilage such as I have described.

The last subject I shall refer to is tubercular epididymitis. It was suggested to me by a man recently in the hospital, and here to-day, whose case was remarkable in one respect, viz. that he had both testes retained just outside the abdominal rings; I have never happened to come across tubercular disease of a retained testis before. The man is forty-two: his father, mother, and one brother, died of phthisis. He himself has had a great deal of chest trouble during the past five years, and two months ago he had hæmoptysis to one pint. He has had glands removed from the left posterior triangle and from the supra-sternal notch. He has been married four years, and his wife has not been pregnant. In February, 1900, he noticed a swelling in his groin, and soon an abscess burst, low down in the fold of the groin, and continued discharging until I saw him in December. When I saw him I thought that I could trace a connection between the sinus in the groin and the epididymis of the retained testicle, which could be obscurely felt as a small body just outside the abdominal ring. The right vesicula was swollen, so I made the diagnosis that it was tubercular disease of an undescended testis. On cutting down and excising the sinus I found that this was the case. The globus minor contained an abscess, whence the sinus started. The body of the testis, though not much bigger than a sparrow's egg, was soft everywhere; the epididymis was, as usual, loosely attached to it. I therefore excised the epididymis, leaving the body of the testis. The external oblique aponeurosis was divided to the level of the internal ring, and the vas cut at the pelvic brim. I show you the specimen. The vas is obviously infected where cut. There is a marked swelling of the epididymis, not very cheesy yet. Under the microscope you will be able to see plenty of tubercular foci in the sections which have been made from it. As to the patient, the scar is normal. You can just feel a small soft rounded body over the external ring:

Digitized by Google

that is the body of the testis. *Per rectum*, I find that the vesicula is still swollen, but less than it was.

I have one other case of epididymectomy for tubercular disease. I excised what I show you from a young clergyman, who had previously been castrated on the other side. Here is the skin of the scrotum and the orifice of the sinus leading down to an abscess in the centre of this huge epididymis. You see the vas is treble the normal thickness. I want you to notice that a complete excision of the epididymis, and of much of the vas, has been done; yet the body of the testicle left behind remains of full size and healthy.

I have shown you these cases to point out the value of epididymectomy. A patient with well-marked tubercular disease of the testicle probably has a worthless testicle from the point of view of procreation, because the vas is blocked. From this point of view, removal of the epididymis does not place the patient in a worse condition than he was before the operation. If you can tell the patient that you will leave the testicle, he will probably consent to operation earlier than he would to castration; and the result will be that, whatever the effect of the internal secretion of the testicle may be, that secretion will be maintained, though no excretion can occur. The operation which you have chiefly to contrast with epididymectomy, however, is not castration, but erasion, *i. e.* incision of the epididymis and scraping it out. Obviously that must be a very imperfect operation as compared with epididymectomy. Epididymectomy may be insufficient. Although it is a great deal more radical than erasion, it may nevertheless be obviously insufficient to rid the patient of the disease; for on getting down on to the testicle you may find that the body is enlarged, or that there are hard masses in its substance, and castration may be required. But it is a better operation than erasion,—one which will lead to union by first intention, and which is far more likely to rid the patient of the disease. Therefore, it seems to me, it is an operation which we ought to use more than we do. The question which required decision was whether it was possible to remove the epididymis and still preserve the testicle. At first one would think that the testicle would atrophy, either from loss of its duct, or interference with its blood-supply; but this does not happen. Sometimes in development the testis and its duct are not connected; but the testis develops normally, and the man is virile. Probably that was a reason which led people to think epididymectomy possible. Vasectomy leads to atrophy of the prostate, but not of the testicle. When I did my first operation, not knowing of any similar operations, I was in doubt as to whether I should be able to remove the whole epididymis and the vas, and leave the testicle its blood-supply. As a matter of fact, it proved easy to remove the epididymis and vas, dividing branches which they receive from the spermatic artery, but leaving the trunk of this vessel which runs to the mediastinum testis mesial to the vas. The clergyman was operated upon three years ago, and I kept sight of him for nearly two years; the testicle remained a thoroughly well-developed organ. None of the changes said to occur after double castration were noted. I think, therefore, that epididymectomy has many advantages, and it is well worth bearing in mind in these cases.

Here is a case of tubercular epididymitis for you to examine: a boy of sixteen, with a sinus leading down to a much-swollen epididymis; the vas is thick to the external ring, and the vesicula distinctly swollen. The right testis is normal; the health good. I shall endeavour in this case to perform an epididymectomy; and I shall warn the patient that recurrence upon the right side is likely, so that, should it take place, he may apply early enough to ensure the possibility of an epididymectomy being done.

Note.—Epididymectomy was contra-indicated by the discovery of marked induration in the situation of the mediastinum testis. Castration was done, and tuberculosis of the body found.

Chloretone in Dusting Powders.—The use of chloretone in dusting powders for painful granulation wounds has been wonderfully successful as far as analgesia is concerned. It does not seem to delay union or granulation, and it can be used in combination with almost any dusting powder. It cannot be said to hasten granulation; all it does is to mitigate or entirely allay pain in the majority of cases.—E. Hollingsworth Siter (*Ther. Gaz.*, March 15th, 1901).

Digitized by Google

PRESENT DAY SKIAGRAPHY.

WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL.

DEMONSTRATION I.

GENTLEMEN,—On Professor Röntgen's brilliant discovery being made known, it was at once clear that the use of the X-rays would be invaluable to the surgeon, but it was not as obvious at first how they would be of assistance to the physician, and this is what we should naturally have expected. Of course the rays show at once the presence of a broken or dislocated bone. A considerable amount of time and work had to be devoted to the subject, however, before we could speak with any confidence as to the significance of the shadows cast by the contents of the thoracic and abdominal cavities. Physicians, however, are beginning to recognise the use of the Röntgen rays in the diagnosis of visceral disease. In writing to the 'Lancet,' October 15th, 1898, I said:—"I venture to assert that a systematic application of the rays to the diagnosis of chest disease will prove of great value to the physician." In an article in the 'British Medical Journal' of January 26th, 1901, we read as follows:—"It really seems as if, when the cost of the apparatus is brought to a reasonable amount, the use of the Röntgen rays in the way described by Dr. Clifford Beale and Dr. Hugh Walsham may become widely applicable in practice as a supplement to other means of clinical diagnosis. The method also seems to bring a little nearer the likelihood of being some day able to satisfactorily treat internal tubercle with chemical rays, as external tubercle (*i. e.* lupus) is being treated to-day with such conspicuous success." It will be my endeavour in this and the following demonstrations to describe as clearly as I can the methods made use of and the uses of the rays in the clinical diagnosis of thoracic disease. First of all it will be well to describe shortly the apparatus and methods employed in taking a skiagram of the chest. An attempt to obtain a picture of the chest that shall be really useful in diagnosis with inefficient apparatus is worse than useless, and only brings the whole method into discredit. The apparatus made use of for exciting the tube are principally two—the coil, and the influence machine, each of these having advantages and disadvantages. To take the coil first, it must be of a certain size. It must be capable of giving a spark not less than twelve to fourteen inches in length. Of course if the physician has the opportunity of making use of the direct street mains and the various modern forms of contact breaker so much the better; but very useful work can be turned out with the ordinary Ruhmkorff's coil fitted with the usual platinum break. The disadvantages of the coil are principally found in screen work. When using a coil with the ordinary break, the flourescence of the tube is very flickering, and it does not light up with the steady flourescence which can be obtained with the influence machine. But, on the other hand, the influence machine is very costly and very cumbersome, and takes up a considerable amount of room, and, of course, it is quite impossible for the physician to take it with him should he be called upon to use the apparatus at the patient's own house. The advantages of the influence machine are the steady flourescence of the tube, and the absence of accumulators, which require re-charging occasionally. The coil made use of in taking the accompanying skiagrams is one giving nominally a twelve-inch spark; but when worked at high pressure it is capable of giving a spark fourteen inches in length. The secondary coil has some fifteen miles of wire wound in seventy sections. It is worked by two four-cell accumulators of twenty-one ampère hours capacity, and an electromotive force of sixteen volts.

I will now come to the tube. The tube made use of in taking a skiagram of the chest must be one of, technically speaking, sufficient hardness, that is to say, of high vacuum or penetrative power. The next most important points to attend to are the time of exposure and the distance of the tube from the photographic plate. The time of exposure must necessarily depend a good deal on the patient; age, sex, muscular development, fatness or thinness of the patient, must all be taken into account to obtain a successful result. Young patients and females will require a shorter exposure than adults and males. Very fat or muscular persons will require a longer exposure than thin and emaciated individuals. As a rule, for thin adults I should say, working with the apparatus I have just described, one minute fifty seconds will probably be sufficient; for a very

Digitized by Google

muscular patient three, or even four minutes may be required, always provided that the coil is of the size described. With regard to the height of the tube above the photographic plate, a more stringent rule can be laid down. We must always remember that the nearer the organ to be skiagraphed is to the photographic plate the sharper

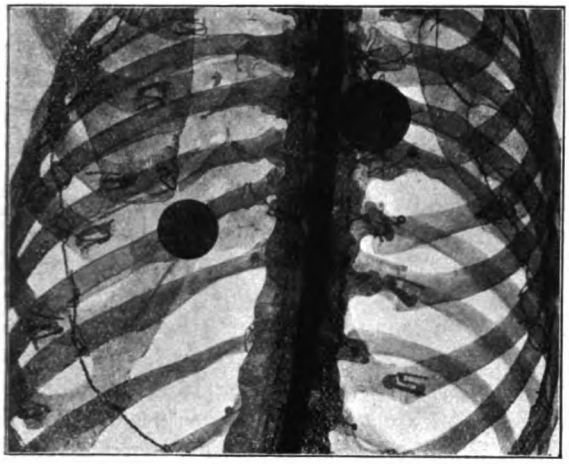


FIG. 1.—Coins on thorax of skeleton, showing the magnification of the one farthest from the plate. The thorax is viewed from the back.

will be its outline, and the less its magnification, and *vice versa*, the further the organ is from the plate the more blurred will be its outline and the greater will be its magnification, the tube remaining immovable. All these points, must be duly considered before drawing any conclusions from the picture, especially when working with the screen, in which case the tube must be placed only a few inches from the chest wall. In taking a skiagram, however, after a certain distance is passed the height of the tube becomes a matter of less importance, as the following illustration will show. Two coins of equal size were placed on the thorax of a skeleton, one on the posterior portion of the ribs near the spine, and the other on the edge of the sternum which was furthest from the plate. The shadow of the coin on the ribs,—that is the one nearest the plate—appears its natural size. The coin on the sternum is magnified somewhat, its diameter being about three eighths of an inch larger than the other coin; the tube was placed four feet above the photographic plate. With the tube only a few inches from the chest wall as in a screen examination, the shadow of the coin on the sternum would, of course, be much more magnified, *e.g.* nearly four times. Of course this magnification of the thoracic viscera becomes of very great importance, because if not taken into account very erroneous notions might be formed of the size of organs, such as the heart, or an aneurysm of the thoracic aorta. Allowing then for this magnification, a good height in chest work is four feet above the surface of the plate. One word with reference to the photographic plate; nearly any make of rapid gelatine dry plate will do, only the more rapid the better. A good size for chest work is ten by twelve inches. I will now pass on to the methods of examining the chest with the X-rays. They may be divided primarily into four principal divisions:

1. Examination by the screen or radioscopy.
2. Examination by the plate or radiography.
3. Examination by the stereoscopic method.
4. Examination by Dr. Guilleminot's method of kinematoradiography.

We may again subdivide these into—

(*a*) Examination from the front of the chest.

(*b*) Examination from the back of the chest.

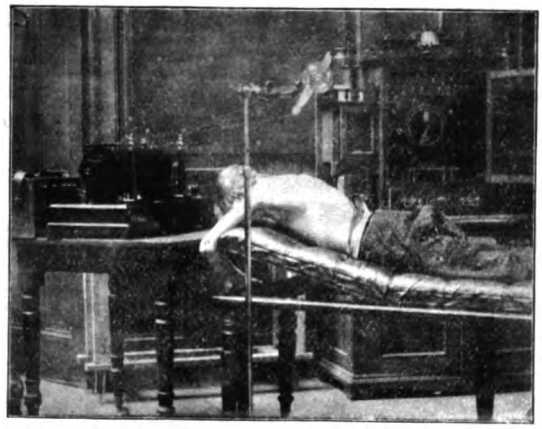


FIG. 2.—Patient in position for skiagram of chest. The dark rim seen under the chest is the plate in the opaque envelopes.

(*c*) Examination by the lateral oblique method, right or left, anterior or posterior, as the case may be.

Perhaps the accompanying illustration will do more than many words to show the method

Digitized by Google

generally employed in making a chest examination. The patient lies on a couch in the prone or supine position with the chest resting on the photographic

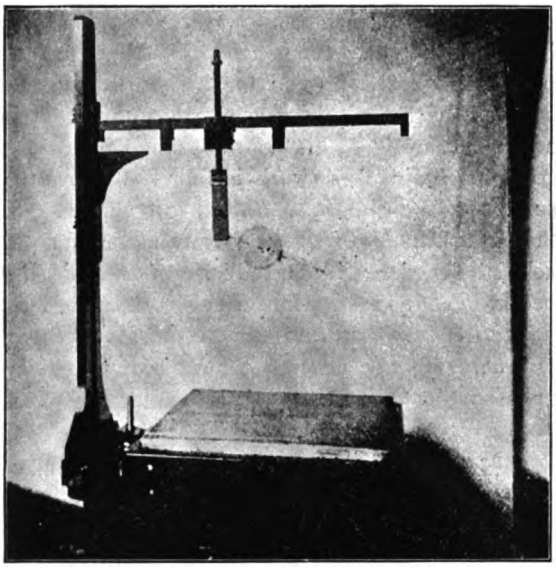


FIG. 3.—Mackenzie Davidson's stereoscopic stand, showing tube on graduated bar with adjustable stops and parchment-covered box. To the left is seen the screw of the lever to fix the plate in position.

plate enclosed in the usual opaque envelopes, and resting on a board to prevent breakage. The tube in a suitable holder is then placed four feet above the surface of the chest of the patient. The above illustration shows the coil, accumulators, and patient in position, and the tube in the holder, as used in the electrical department of St. Bartholomew's Hospital. The anti-cathode or anode of the tube must be made accurately at right angles to the plane of the plate. I usually employ the anterior method myself, because this method gives a sharp outline of the heart, but of course both may have to be used, especially in examining the chest for a suspected aneurysm; the reason for this I shall return to later on.

To obtain a stereoscopic view of the chest the procedure is somewhat different, but this method should be always employed, especially in the case of a suspected aneurysm. We require a special apparatus, such as that invented by Mr. Mackenzie Davidson. It consists, as seen in the above illustration, of a stout wooden frame covered with parchment; an upright rod carries a horizontal graduated bar, on which the tube slides. This horizontal bar can be raised or lowered as desired: the patient rests with the chest on the parchment covering. The plate is then placed under the parchment and kept in position by means of a lever and screw; the horizontal bar is now placed at the required height above the surface of the chest and the tube is brought to the zero point, which is at the middle of the graduated bar. The tube in this way is brought vertically over the spine of the patient. It is now displaced thirty millimetres to the right of the zero point, and an exposure given, say of one minute fifty seconds. The current is then switched off, and the plate is removed and another one placed in position without disturbing the posture of the patient. The tube is now displaced thirty millimetres to the left of the zero point and another exposure given of the same time as the former. We have thus obtained two pictures of the chest from two different points of view sixty millimetres apart. When prints are taken from these negatives and placed in a Wheatstone's reflecting stereoscope, a stereoscopic effect is, of course, obtained. By this method we obtain the third dimension in space, which must obviously be of very great importance in

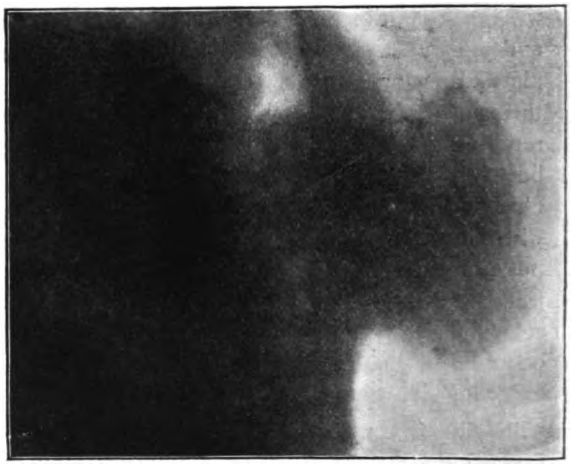


FIG. 3A.—Chondro-sarcoma of sternum. The growth is seen penetrating the sternum and partially filling the retro-sternal triangle. Taken by lateral oblique method.

examining a chest for an aneurysm. By this means, also, we may be able to localise an aneurysm to the part of the vessel from which it originates.

Digitized by Google

The process may seem a complicated one, but in reality it is extremely simple. The stereoscopic method can be also applied with the screen after the very ingenious method of Mr. Mackenzie Davidson.

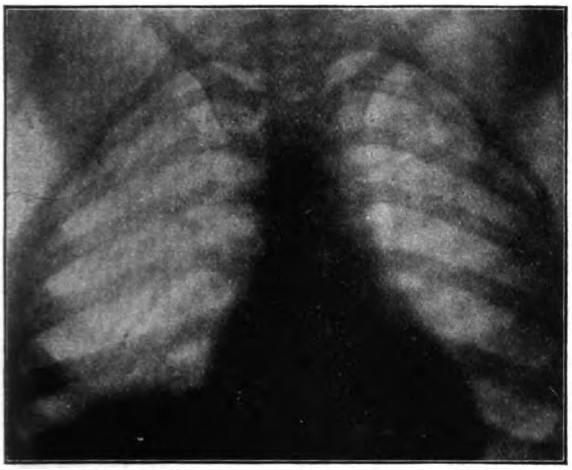


FIG. 4.—Shadows seen in a normal chest. In addition to the bony framework is seen the shadow of the heart and diaphragm, the cardiac lines, the aorta, and the shadow of the axillary folds.

I will now proceed to describe the third way, that is the lateral oblique. This is a method used by M. Mignon, of Nice. It may be used either with the screen or with the plate. It is well known to all workers in chest radiography and radioscopy that a strictly lateral view of the chest shows nothing definite, but if an oblique view can be taken much may be learnt of the condition of the mediastina. On the screen one sees a clear zone of somewhat triangular form bounded as follows: below by the convex shadow of the diaphragm, in front by the shadow of the heart, and behind by the vertical shadow of the vertebral column, which shadow, of course, includes those of the aorta and œsophagus. M. Mignon proposes to call this clear triangular zone the retro-cardiac triangle. There is another somewhat clear triangular space in front of the heart shadow corresponding to the anterior mediastinum. This may be called the retro-sternal triangle. Tumours or enlarged glands in the mediastina, anterior or posterior, can occasionally be well seen by this method of examination. Any bulging of the vertical spinal shadow would indicate an aneurysm springing from the descending thoracic portion of the aorta or tumour of œsophagus. I have tried this method in a few cases; the anterior or retro-sternal triangle is seen clearly enough, but the shadows in the retro-cardiac triangle are much more difficult. I have seen this retro-cardiac triangle, however, filled up with shadow in a case of enlarged mediastinal glands (see Fig. 3A).

My subject naturally falls into two principal divisions: (1) the examination of the lung and pleura, and the (2) examination of the heart and great thoracic vessels with the contents of the anterior and posterior mediastinum. Now it is quite obvious that it would be perfectly impossible to say whether a cardiac murmur were present or not without a knowledge of the normal heart-sounds, so in exactly the same way it is quite impossible for an observer to give an opinion as to the presence of any abnormal shadow within the chest without his being familiar with the shadows caused by the contents of a normal thorax. I shall, therefore, first describe shortly the shadows seen in a perfectly normal chest. Fig. 4 shows a skiagram of a perfectly healthy chest; the shadow caused by the heart or, more accurately speaking,

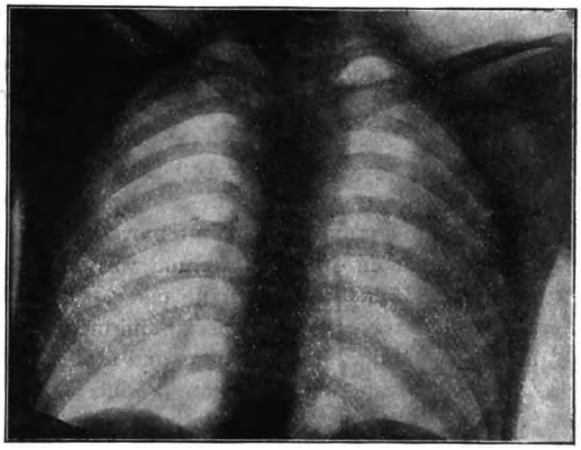


FIG 5.—Skiagram of chest with lungs and heart removed It shows the cardiac and mediastinal lines and the shadow of the aorta.

the shadow cast by the pericardium with the inclosed heart, of course is at once obvious. The shadow of the aorta may be traced nearly up to the manubrium sterni, where it is lost. Now in a normal chest, to the right and to the left of the

Digitized by Google

heart's shadow we see certain lines or streaks, which, for the want of a better name, we may call the cardiac and mediastinal lines. These lines are seen in all normal chests, and it required a great deal of work and thought to determine what part of the thoracic contents causes these shadows. I endeavoured to clear up this point in the following way. I took a skiagram of a body (which had died with a disease other than cardiac) on the post-mortem table. I then removed the sternum, opened the pericardium, and removed the heart. The sternum was then replaced in position, the integuments closed over and another skiagram taken. The sternum was again removed, and both lungs were taken out; the sternum was again replaced, and another skiagram taken. By this means we see (Fig. 5) these cardiac and mediastinal lines still remain in the skiagram, and I think there can be no question that they are produced by the junction of the visceral pleura with the pericardium. Of course these lines in the living chest do not come out with anything like the distinctness which is shown in the accompanying illustration, because they are in constant movement, caused by the systole and diastole of the heart. On looking again at the skiagram (Fig. 4) it will be seen that there are two broad shadows one on each side of the chest, extending upwards and outwards from the sternum towards the axilla. These shadows by the stereoscopic method are seen to be outside the thorax altogether, and are produced undoubtedly by the anterior axillary folds. The shadow of the diaphragm with the liver below is of course at once obvious. The shadow caused by the spleen is not at all definite, and sometimes we cannot see it; but frequently below the diaphragm a clear zone is recognised, which probably corresponds to the space between the cardiac end of the stomach and the shadow caused by the spleen. Of course the skiagram of the healthy abdominal cavity is very disappointing, with the exception of seeing the before-mentioned shadow of the liver, and this light zone corresponding somewhat to the position of the spleen, nothing is seen but the shadow of the spinal column, provided, of course, that the abdominal viscera be healthy (see Fig. 6). The only abdominal disease that comes under the care of the physician that I shall have occasion to mention is renal calculus, to which I shall return later in my subject. There are two more shadows to be observed in the healthy thorax, and they are the edges of the scapulæ on each side. It is as well, if possible, to obliterate these shadows, because they may be somewhat confusing in examining the chest for a pleural effusion. This can be done by taking the patient in the prone position with arms extended and hanging over the edges of the couch, as seen in Fig. 2. In this position the scapulæ are turned outwards, and their shadow is thus removed from the photographic plate. One word more about

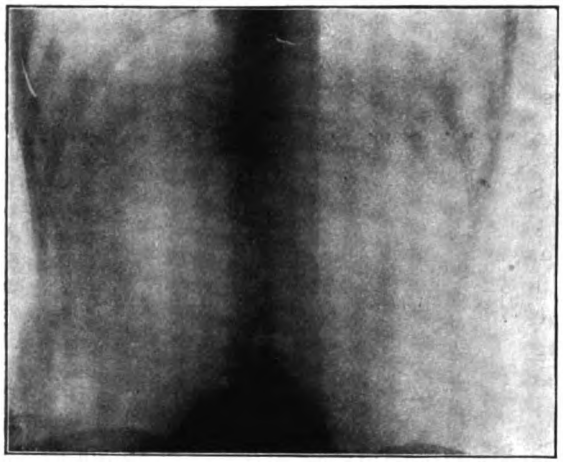


FIG. 6.—Skiagram of healthy abdomen. It shows the spinal column, the three lower ribs, and crests of the ilium.

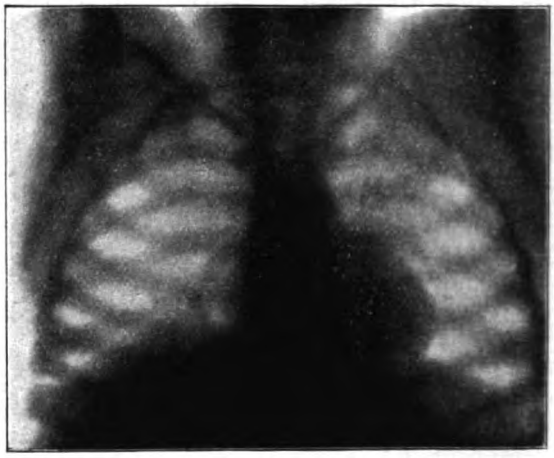


FIG. 7.—Normal chest of child, showing high position of the diaphragm and consequently more horizontal position of the heart. It also shows well the cardiac lines to the right of the heart shadow.

Digitized by Google

the normal chest. Does the skiagram of the chest of a child differ in any way from that of the chest of an adult? It does in two important points: one is the much more horizontal position occupied by the shadow of the heart in childhood, and the other is the shadow of the diaphragm stands much higher in the child than in the adult. The healthy lungs, of course, are perfectly transparent to the rays from apex to base, as seen in the above illustrations. This is a very important point to bear in mind.

Treatment of Puerperal Fever.—In the treatment of puerperal fever one must first enjoin perfect rest, and search for the seat of infection. If the latter is then found to be located about the vagina or vulva, the part should be wiped with absorbent cotton moistened with some antiseptic solution that is not caustic, and then dusted with aristol or some similar antiseptic powder. Where the seat of infection is covered with a greyish-white membrane the case is more serious than where there is simply a free pus-formation. If no infection can be found about the entrance to the genital tract, the vaginal mucous membrane and the vaginal portion of the cervix should be minutely inspected, care being taken not to reopen small wounds. If the examination is still negative, and the patient's condition good, the physician should wait and watch, remembering that the retained placental tissue is often soon expelled spontaneously. If, however, her condition is not good, the interior of the uterus should be examined under strict antiseptic precautions. The use of the curette in cases of septic endometritis is strongly opposed. An intra-uterine douche should be given both before and after the intra-uterine manipulation, and ordinarily it is better not to repeat this douche. The exception to this rule is found in those cases in which the temperature remains high and there is a free purulent discharge from the uterus. If the infection is localised in the pelvic cellular tissue or the pelvic peritoneum, the ice-coil and inunctions of Credé's ointment of metallic silver will be beneficial. H. J. Boldt ('Boston Med. and Surg. Journ., February 21st, 1901).—*Monthly Cyclopædia of Practical Medicine.*

FOUR CLINICAL LECTURES
ON
SOME AFFECTIONS OF THE KIDNEY.

Delivered at Westminster Hospital.

By A. H. TUBBY, M.S., F.R.C.S.

Lecture III.

Gentlemen,—To-day we will consider tubercular disease and some tumours of the kidney. With regard to the former, we must clearly understand what we mean by it from a surgical point of view. It is important to exclude miliary tubercular disease of the kidney, that is to say, such a condition as is found when acute tuberculosis breaks out, and which shows at the post-mortem, an event often only a short time after the onset of the illness, a large number of grey miliary tubercles scattered about the kidney. This is not tubercular kidney in the usual acceptation of the term. Tubercular disease commences in the cortex, or in the medulla, or both; it is particularly prone to affect the Malpighian pyramids and the cortex, leaving the columns of Bertini partially intact, so that we find the kidney substance eaten out in wedge-shaped cavities. If one examines an advanced case of tubercular disease of the kidney, there are several of these wedge-shaped suppurating spaces, varying in number from one to seven or eight. The reason that the kidney takes this appearance is because the most resistant parts to tubercle are the columns of Bertini. At the same time excessive changes go on in the capsule, which becomes extremely thickened, and often indurated. The neighbouring tissue and the perinephritic tissue are affected, and these may become so hard and dense that one can only with difficulty cut through them, so that it is often difficult to find the kidney through the thickened surroundings. If the disease goes on for a sufficient length of time the cortex becomes converted into a hard fibrous india-rubber-like material. At the same time ulceration of the pelvis of the kidney ensues, and the disease extends downwards. This is a very striking clinical point about the disease, for it spreads down the urinary tract, invading it at considerable distances from the original infection, and it makes its way into the ureter, and thence

Digitized by Google

into the bladder, urethra, and vesiculæ seminales. Dr. Hilton Fagge has described a case in which there was ulceration at the junction of the pelvis of the kidney and the ureter. The ureter is sometimes blocked, so that there may be a temporary condition of hydronephrosis associated with tubercular kidney. I saw a post-mortem examination of one case in which the disease had begun in the left kidney, extended down the left ureter, crossed the trigone of the bladder, reached the right ureter, and was in process of extension upwards from there. That was a very striking case, and is in itself a good reason for operating upon a tubercular kidney. There is another very good reason, viz. it sometimes happens that one kidney is affected, and is the primary site of the tubercular deposit. If this were not so it would not be right to bring tubercular kidney within the realm of surgery.

With regard to the contents of these cavities in the kidney, you find, as a rule, that they are covered by cheesy matter, which is semi-fluid ; but when the disease has existed for a long time you find the walls of these cavities have become smooth. I believe that to be the condition of the patient in Hollond Ward. She has tubercular kidney, with two or more large cavities, and on inserting one's finger into the purulent cavities one finds the walls very smooth, and very much like an old cavity in the lung.

The kidney has since been removed, and this supposition verified.

The symptoms of tubercular disease of the kidney are at first insidious. The patient becomes out of health, and complains of general malaise, and gradually gets weaker. Perhaps also you will find at this stage a slight rise of temperature at night ; he has a bad appetite and loses weight. Shortly after the onset the patient begins to complain of pain. This is generally quite different from that of renal calculus, though by no means always. The pain is in the lumbar region, and probably the most tender spot is at about half an inch below and outside the tip of the last rib. The character of the pain is dull and aching, but occasionally it becomes paroxysmal ; and these paroxysms may be due to blockage of the ureter by some caseous material, resulting in distension of the kidney. Late in the disease there are signs of tubercle elsewhere. For instance, in the case in Hollond Ward there are some signs of tubercle at the apex of the right lung.

The signs which you notice are as follows : First of all there is a renal tumour. I use the word "renal" here in a descriptive sense. It is a swelling starting in the lumbar region, and it can be felt from the back ; it grows forwards, and not backwards. It does not move on respiration unless it is extremely large, and it has colic resonance in front of it. I have spoken to you before about colic resonance. The tumour, as a rule, is hard and uniform, but varies in size, and is occasionally fluctuating, as in the case of the patient in Luke Ward. Next, there is the typical tubercular or hectic temperature. It has often been asked, "What is the cause of hectic fever? It is supposed that it is due to the absorption of pus-serum or toxin, which sets up an alternate fall and rise of temperature. As a rule the urine is acid, except in one condition, namely, in cases where the disease may have extended to the bladder. Then the urine, which remains in the bladder and becomes decomposed is ammoniacal. Certainly in the earlier stages of the disease the urine is acid, as it is in the case of calculous pyelitis. The amount of urea is lessened because the secreting substance is lessened. We had a striking feature in the case in Luke Ward, namely, that before the exploratory operation, and before we interfered at all with the kidneys, the amount of urea varied from 200 to 300 grains a day. When the pus had been let out of the diseased kidney the amount of urea increased to something like 400 grains a day. It is difficult to explain this, except on the supposition that the secreting substance which remained in the diseased kidney was unable to do its work because of the pressure of pus before the operation, and that when this was relieved the blood could flow freely through the remainder of the kidney, and so the amount of urea was increased. It is very important indeed for you to always take a note of the amount of urea which is passed. If it be reduced to a half it is not, as a rule, advisable to operate, because operation interferes with the secreting substance of the kidney, and you have no means of ascertaining how much urea is coming from the other kidney. Some day it may be discovered how much urea comes from one kidney, and how much from the other. Several attempts have been made to find that out.

Digitized by Google

but they have not succeeded. A German practitioner used to go down to the post-mortem room at Guy's and pass an ingenious instrument. He said he could get his instrument into a ureter, so that he could draw off the urea from it, and ascertain the amount of urea secreted by that kidney. Such a thing might be possible in the female, but as to the male, "I hae my doots aboot it." In the male the orifices of the ureters into the bladder are too small to admit of one being certain of passing an instrument into them through the urethra. Certainly unskilled attempts in the living subject might very readily result in perforation. If the amount of urea in the urine be lessened, the specific gravity of the urine is also lessened.

From time to time there is blood in the urine, but to nothing like the same amount as in stone, or in malignant disease. The blood is closely mingled with the urine, and as a rule appears in it only at long intervals. The urine itself is thick with pus, and the flow of pus is constant and copious. The points to note are little blood and much pus. In stone there is much blood, and at first little pus; later on there is much blood, but not so much pus as in tubercle. In the urine is found a large amount of caseous material and *débris* and connective tissue, and in this you will find elastic tissue. The addition of acetic acid to the deposit in the urine clears the opacity, and makes the yellow elastic fibres more prominent. If you still have any doubt about there being yellow elastic fibres present, you should boil the urine with caustic potash, when the elastic fibres will not dissolve, but the rest will. Occasionally in the urine of these patients there are tubercle bacilli. I think these are found in two cases out of six. You should centrifuge the urine, and then carefully examine a number of slides taken from the deposit. To look at one slide is insufficient; you should examine a large number of slides before you conclude that there are no tubercle bacilli. Should your examination prove futile, take some deposit, add a little disinfectant to it, and inject some of it into a guinea-pig. In many cases there will be a tuberculous reaction.

These patients also suffer from irritation of the bladder; they have rigors and sweating, and towards the end of the illness there are the symptoms of phthisis, loss of strength and flesh, and diarrhœa, with vomiting.

Now with regard to the complications. You often find the deposit of tubercle bacilli elsewhere, especially in the lungs, in the brain—these patients often dying from tubercular meningitis,—and local deposits in the urinary tract.

As to the *diagnosis* of tubercular kidney, this is admittedly very difficult. This condition is frequently mistaken for stone, and stone is frequently mistaken for tubercle. The points upon which you are to rely for the diagnosis are the history, the presence of pus in acid urine, caseating tubercle and connective tissue in the urine, and tubercle bacilli. Lastly, it is quite justifiable to make an exploratory operation.

With regard to *treatment*, medical treatment may be regarded as valueless. Cod liver oil will not touch the kidney. Surgical treatment is the only procedure, and it may be on two lines, either by cutting into the kidney (nephrotomy), which is of no permanent value, or nephrectomy. You may have to do a nephrotomy merely to evacuate pus and give relief from pain, as I had to do in the case of Mrs. H—, in Hollond Ward. She was in too feeble a condition to admit of nephrectomy; it would have been fatal. Her tumour is now smaller, she is relieved, and her temperature has fallen. But that measure is not curative. The indications for merely cutting into the kidney are as follows:—If the patient is suffering from pent-up pus, if there is much pain, and if the general condition of the patient is bad. All these conditions were present in the case in Hollond Ward.

Nephrectomy is, of course, a more difficult and serious matter. The indications for it are as follows:—First of all you should only do it in early cases, and if you are quite sure of your diagnosis. Secondly, when the amounts of urine and urea passed per day are nearly normal. Thirdly, if the other kidney can be felt not to be enlarged or tender, showing that in all probability it is not involved. Fourthly, if the general condition is good. The remote result is fairly satisfactory, but you must not hope for too much from the operation, on account of these patients so often becoming the subjects of tubercle elsewhere in the body.

We now come to tumours of the kidney. There are the congenital tumours, such as sarcoma, cystic disease, and nævoid growths. Then there are those of acquired origin, as hydatid cysts, cystic disease, lipoma, fibroma, adenoma, sarcoma, carcinoma,

Digitized by Google

and secondary growths. It is a strange thing that you find sarcoma in infants before birth, and in children up to five or six years of age. Then there is a lull in its recurrence, and it becomes very rare until from thirty to forty years of age. So one may say that sarcoma of the kidney is comparatively common up to the age of five, and between thirty and forty years of age. Cystic disease may be either congenital or acquired. If it is acquired it is very often associated with contracted granular kidney. The origin of the cysts is that certain of the tubules become contracted at intervals by fibrous tissue, and then they dilate, assuming the appearance of cysts. Nævoid growths of the kidney are curiosities.

We had an example of cystic disease of the kidney two years ago. The man had a huge cystic kidney. We ascertained by an abdominal incision that the other kidney was unaffected, and removed the cystic kidney. It weighed two to three pounds, and, of course, was rather difficult to get away.

Cystic disease may be either congenital or acquired. The kidney is transformed into numerous cysts, the contents of which are pale straw-coloured fluid. Such a kidney may attain to an enormous size. It is painless, irregular, semi-fluctuating, and, most important, there are no signs in the urine, no blood, and no pus,—in fact, nothing beyond the diminution of urea.

The treatment is by nephrectomy, but only in certain conditions :—(1) If the tumour is of very large size, and (2) if only one kidney is involved.

Sarcoma affects the kidney, and is present usually in young children from one to five, and in adults at about forty. If the sarcoma is of congenital origin, it may attain such an enormous size that the child is taken away from the kidney instead of the kidney being taken away from the child, and these cases are fatal. The type of sarcoma is either the spindle-celled or the round-celled, and the striking point about sarcoma in the kidney is that it very rarely invades the surrounding tissues. It does not extend to the diaphragm nor into the perinephritic tissue nor to the muscles, but it does extend along the vessels. The renal vein rapidly becomes blocked, and so does the inferior vena cava, and there are malignant infarcts in the lungs. As a rule only one kidney is affected, and it is very rarely that there is an extension of growth down the ureter.

The type of carcinoma met with in the kidney is encephaloid, or soft carcinoma. It affects adults over forty, and grows very rapidly indeed.

The symptoms of malignant growth of the kidney are, firstly, pain, which is constant and of a radiating nature. But it sometimes happens that it is entirely absent, although the tumour can be felt. Secondly, hæmaturia is fairly constant. The amount of blood varies. In the intervals of the flow of blood from the kidney the urine is quite normal. But hæmaturia is not by any means present in all cases. In 50 per cent. of the cases there is malignant disease of the kidney without pain and without hæmaturia, and nothing can be felt except the growth in the loin. These people frequently suffer from nausea and vomiting, and, of course, undergo gradual exhaustion.

Now as to the signs. A renal tumour can be well defined, because you can get your hand all round it, *i. e.* between the last rib and the kidney, between the crest of the ilium and the kidney, and the liver and the kidney. The growth does not extend into the neighbouring tissues, but remains isolated. It is smooth and uniform, and it does not grow backwards, but towards the anterior abdominal wall. Later on there are ascites and œdema of the feet, showing that blocking of the renal vein and of the vena cava has taken place.

The treatment resolves itself into whether the kidney shall be or shall not be excised. The value of nephrectomy is rather doubtful, because the number of deaths after operation is very great, and secondly, the probability of recurrence is greater still. Deaths from sarcoma of the kidney complicated by nephrectomy number at least 60 per cent., and of carcinoma 71 per cent. Therefore one would hesitate before making any attempt to remove the kidney under such conditions. Moreover operation may be followed by secondary growths in the lungs or elsewhere.

In contrast to the foregoing I ask you to remember Abbe's two successful cases in children. He removed a sarcomatous kidney from a child, and it was alive two years afterwards. He removed a similar kidney from another child, which was alive three and a half years afterwards. Those are very successful cases, and exceptionally so. Unfortunately the general result is collapse and failure.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 453. WEDNESDAY, JULY 3, 1901. Vol. XVIII. No. 11.

CONTENTS.

PAGE

*Present Day Skiagraphy. WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL. Demonstration II 161

*With Mr. Lockwood at St. Bartholomew's Hospital. CARDINAL RULES IN DIAGNOSIS 172

*The Practice of Blood-letting. By J. F. BRISCOE, M.R.C.S. (*continued*) ... 174

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

PRESENT DAY SKIAGRAPHY.

WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL.

DEMONSTRATION II.

GENTLEMEN,—I will to-day consider the application of the rays to the diagnosis of diseases of the pleura. A simple dry pleuritis, as seen with the screen or with the photographic plate, is not a striking example of the power of the rays; with the exception of a very ill-defined slight shadow, nothing is noticed. It is far otherwise, however, with pleurisy with effusion, be that effusion serum or pus. In such a case the Röntgen rays not only show that fluid is present in the pleural cavity, but they are capable of diagnosing the kind of fluid, be it serum or pus. I will now give you an example of pleurisy with simple serous effusion.

A little boy, 6 years of age, was sent to me by my colleague, Dr. Clifford Beale, for an X-ray examination of the chest. The case was an obscure one, and the differential diagnosis lay between pleurisy with effusion and a broncho-pneumonia of the left base. There was no heart displacement, that is, there was no apparent displacement; but if we remember that the apex of the heart in a child of his age occupies a position, as shown by radioscopy, further to the left than in the adult, there probably was some slight displacement of the organ to the right. The skiagram shows conclusively, however, that fluid is present in the pleural cavity; on the left side of the chest we see a shadow with a well-defined margin extending from the apex downwards and inwards to the articulation of the seventh rib, at which point the shadow extends from the spine to the ribs. The shadow is a uniform one, and does not obliterate the shadow of the ribs, although it obscures the heart shadow. A clear oval space is noticed between the upper margin of this shadow and the spine, corresponding, no doubt, to the collapsed but not altogether airless left lung. On looking carefully at the skiagram, we see that the slope of the ribs is different on the two sides;

Digitized by Google

those on the affected side being rotated upwards and outwards. On the evidence of the skiagram the chest was tapped, and thirty ounces of serum were withdrawn. Another point worthy of note is

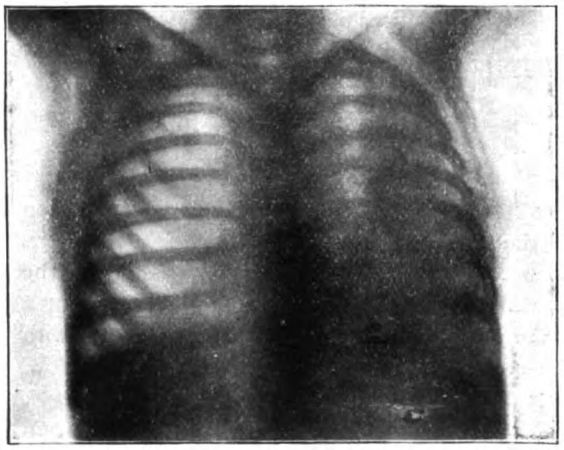


FIG. 1.—Fluid in left pleura. The chest is viewed from the front.

that the skiagram gave no indication of any tuberculous mischief either of the lung or pleura, and the opinion was expressed at the time the chest was examined with the rays that the effusion

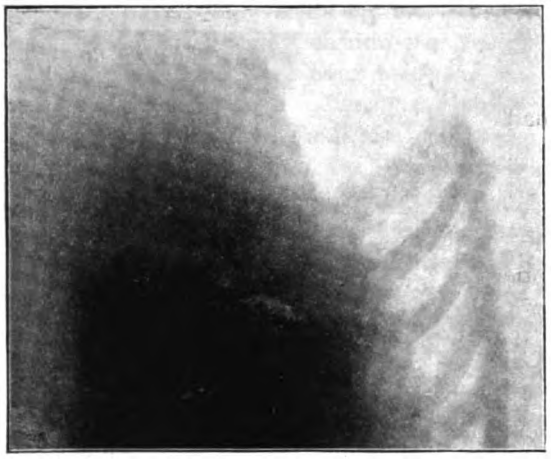


FIG. 2.—Empyema of right pleura. The chest is viewed from the front.

was a simple one. The after-history of the case has fully justified this prognosis.

The next two skiagrams I give as illustrations of empyema.

A lad, 19 years of age, was admitted to the City of London Hospital for Diseases of the Chest with a diagnosis of right empyema. This skiagram was taken before the chest was opened. There is seen to be a very dense uniform shadow occupying the whole of the right side of the chest, and completely obliterating the outline of the ribs; the heart is displaced to the left, the left margin of the shadow being formed by the left border of the organ. The next picture was taken after the

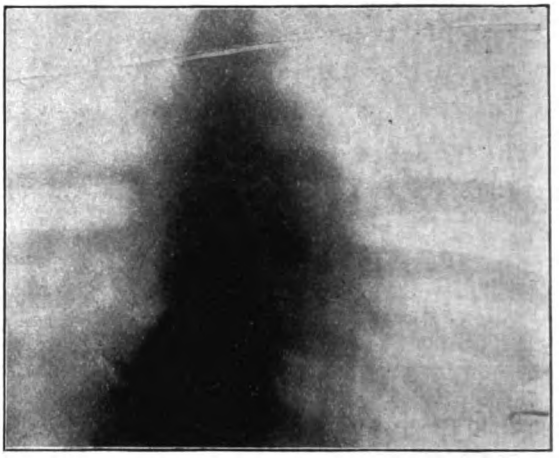


FIG. 3.—Empyema after tapping. Chest viewed from the front. Shadow at right base, probably caused by thickening of pleura.

evacuation of the pus. There is a striking difference to be seen between the two. The heart has returned to its normal position; the compressed lung has expanded again to its normal size, and the pulmonary image of the affected side is almost as clear as that on the unaffected side, but not quite. There is a slight shadow left, due, no doubt, to some thickening of the pleura.

The above cases will, I think, suffice to show the value of the rays in the diagnosis of pleurisy with effusion. The next disease of the pleura which I shall mention is pneumothorax. Here we get with the screen and plate what we should expect, that is, increased brilliancy of the whole of the affected side, as the rays have merely to pass through a large air-containing cavity. There is generally, however, an obscure oval shadow, situated by the side of the spine, due, without doubt, to the compressed and collapsed lung (see Fig. 4). But if this collapse is prevented by dense adhesions, as usually happens in cases

Digitized by Google

of old pulmonary tuberculosis, other shadows may be seen on the affected side crossing at some parts the clear zone. Pneumothorax may be, of course, at once distinguished from emphysema—where we also get increased brilliancy of the pulmonary image,—by being *unilateral*. In emphysema the increased clearness of the pulmonary image is *bilateral*. The diaphragm on the affected side in pneumothorax stands much lower than on the non-affected side, provided, of course, there is a plus pressure in the pleura. The diaphragm in emphysema is lowered equally on each side. In cases of pyopneumothorax we have a dense shadow below with increased brilliancy above the boundary line of the pus. Very dense and very thick pleural adhesions caused a very perceptible shadow on the screen. No doubt calcareous degeneration of old pleural adhesions would throw a very dense shadow, but up to the present I have not had an example of this rare condition.

I now pass on to diseases of the lung, and here the question at once naturally arises, can the Röntgen rays detect really early tubercle in the lung? Speaking from over five years' experience, I should say that the rays can detect tubercle in the lung as early as the stethoscope, or perhaps

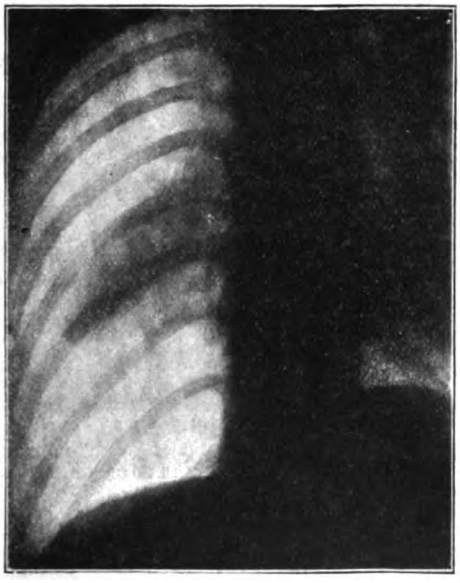


FIG. 4.—Pneumothorax.

earlier; but whether we shall with improved methods be able to diagnose tubercle with certainty with the rays before the stethoscope I think is at present an open question. That both miliary and caseous tubercle cause definite shadows on the screen and photographic plate there can be no doubt whatever. The following skiagram is

FIG. 5.—Miliary tuberculosis of the lungs, taken after their removal from the body. The skiagram shows the tongue, œsophagus, larynx, trachæa, bronchi, and lungs.

that of the lungs of a child with miliary tuberculosis taken after their removal from the body. On looking carefully at the plate we see the shadow of the miliary tubercle scattered through both lungs. The following skiagram is an example of a case of miliary tuberculosis which was first diagnosed by the aid of the X-rays. A child, six years of age, was admitted to the hospital under the care of Dr. Eustace Smith. For some time the diagnosis lay between miliary tuberculosis and enteric fever. The above skiagram shows well the shadow of the miliary tubercle in the lungs. Caseous tubercle again casts a very dense shadow, far more dense than that caused by miliary tubercle, as the following picture will show. The apex of the lung, which at the post-mortem table was seen to be caseous and breaking down, casts a very dense black shadow on the photographic plate, with numerous smaller lighter spaces corresponding to the small cavities that were present. The shadow of the tubercle in the lower lobe of the lung is nothing like so dense.

Take again, for instance, the following skia-

Digitized by Google

gram, which shows the caseous tubercle with cavitation in the lung, and also tubercle in the left kidney from the same case. On looking at the lung we see the shadow of the tubercle

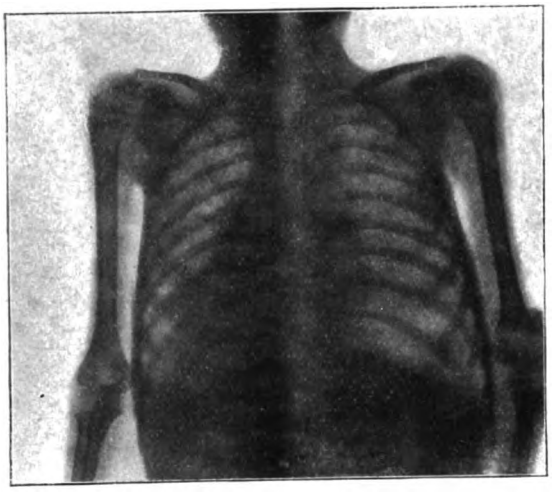


FIG. 6.—Miliary tuberculosis of the lungs. Chest viewed from the back. There is seen a general stippling of both lungs from apex to base.

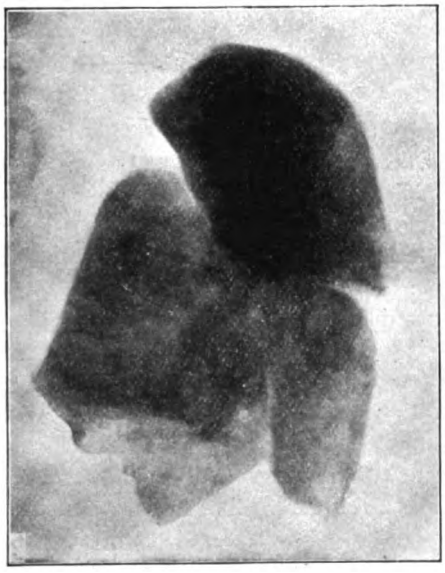


FIG. 7.—Chronic pulmonary tuberculosis of lung; showing the dense shadow of the caseous upper lobe and the much less dense shadow of the grey and yellow tubercle in the lower lobe.

scattered all through the organ, and on looking at the kidney we see distinctly that there are caseous foci in the gland. So much for the lung.

The next skiagram (Fig. 9) is that of a case of pulmonary tubercle skiagraphed on the post-mortem table before the thorax was opened. The left pleural cavity contained about half a pint of serous fluid; the upper edge of the fluid shadow is well shown. We also see the shadow of the tubercle in the lungs; but, of course, not as dis-

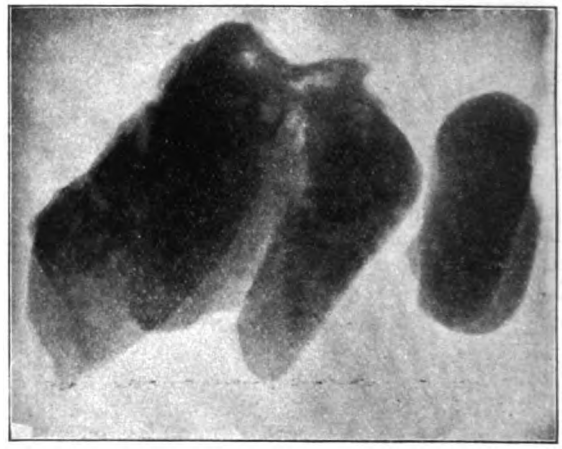


FIG. 8.—Tuberculous lung and kidney from the same body. The lung shows the shadow of the caseous and grey tubercle, with numerous small cavities. The shadow of the small caseous tubercle in the kidney is also seen.

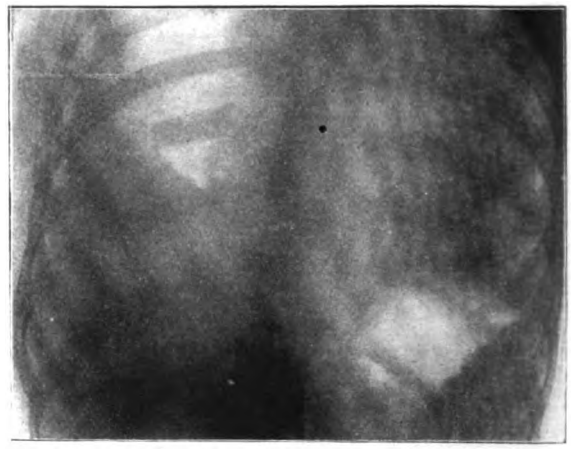


FIG. 9.—Skiagram of chest taken after death, showing fluid in the left pleural cavity and the tubercular shadow on the right side.

tinctly as outside the body, the shadow being somewhat obscured by the muscles and integuments of the chest. It has been suggested that these shadows seen in the pulmonary image are produced by patches of congestion surrounding the tubercle or the tuberculous deposit. I think

Digitized by Google

not. The following skiagram shows a rib, a piece of muscle, and a blood-clot of exactly the same thickness. It will be seen that the muscle and blood-clot cast shadows of almost equal

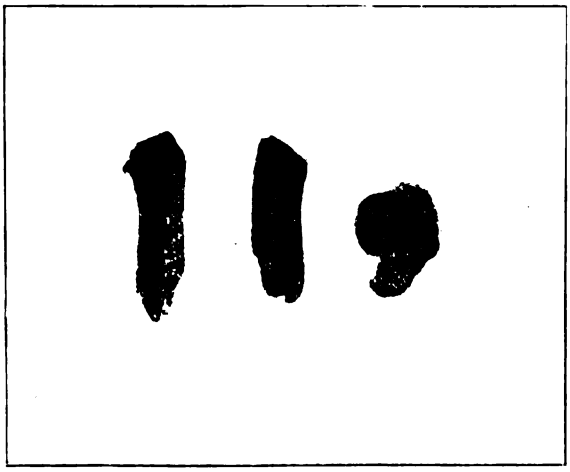


FIG. 10.—Skiagram of bone (rib), muscle, and blood-clot; showing equal density of the shadow of muscle and blood-clot.

density, the bone, of course, being the densest of the three. The exposure, I need hardly add, was equal in all three cases. Again, the statement that blood next to bone is the densest tissue in the body is not borne out by comparative skiagraphy. As the above example shows, there can be no doubt that clotted blood gives a dense shadow; fluid blood, on the contrary, being transparent to the rays, as the following experiment will show. The heart and lungs were removed from the body, and all the vessels ligatured on the one side; on the other side the vessels were allowed to bleed into the pleura. The sternum was replaced, and the integuments closed over. The chest was skiagraphed. It is seen at once (see Fig. 10A) that one side is as clear as the other, proving conclusively, to my mind, that fluid blood is transparent to the rays. In nearly all cases of pulmonary phthisis we find tracks of shadow across the pulmonary image. On what do these tracks of shadow depend? Are they due to congested vessels surrounding the area of tubercular lung tissue, or are they due to cellular accumulations filling the air-cells of the lung? I think there can be no doubt that they are due to the latter, because, as I have shown, blood, certainly fluid blood, is transparent to the rays. If these shadows are not due to cellular accumulations in the air-sacs, it is very hard to say on what they depend.

To show as clearly as possible that the shadows found in the tubercular lung are actually due to cellular accumulation within the air-sacs and not to blood congestion, I performed the following experiment in addition to those above. The lungs were taken from a body which died of phthisis. One lung was skiagraphed in the condition in which it was when taken from the chest. The other lung was first subjected to soakage in water. It was soaked in water for three or four hours, and the lung was then squeezed in a cloth, and again soaked and again squeezed to break up as many of the blood-corpuscles and to wash the lung as free from blood as possible. The vessels were then injected with water, and thoroughly washed out. The lungs were then put on a photographic plate and skiagraphed. I think that we shall see by looking at the illustration (Fig. 11) that the lung shows very little if any difference from the one that was subjected to no washing. The washed lung is marked by a coin. It may be as well, finally, to mention the principal points to be attended to in examining a chest with the

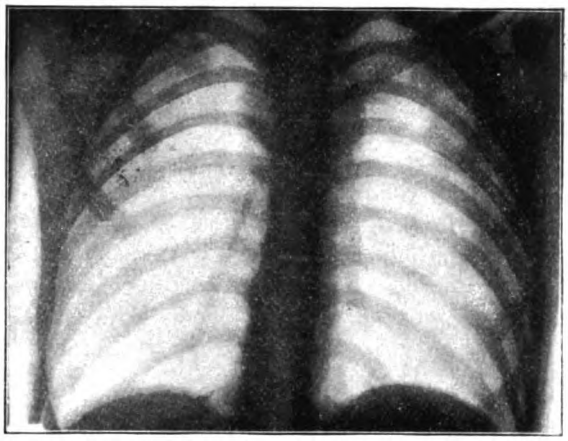


FIG. 10A. Skiagram of chest taken on post-mortem table, with right pleural cavity full of blood.

screen for suspected pulmonary tubercle. The patient should sit, preferably, on a revolving stool, and I think it is as well to begin the examination from the back. The tube is placed about three

Digitized by Google

inches from the front wall of the chest, being on a line drawn from one nipple to the other, and it is better during the examination to keep the tube at this position. The screen being placed

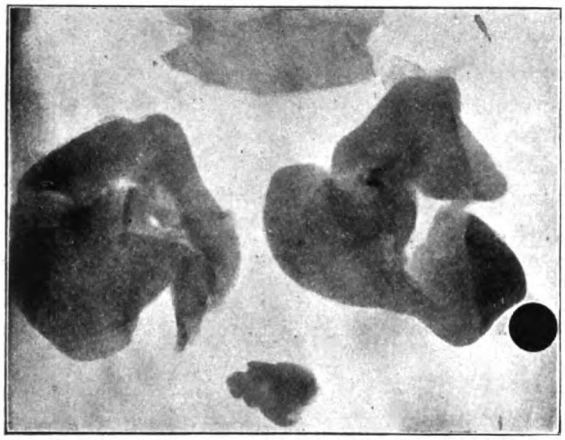


FIG. 11.—Skiagram of lungs. The one to the right was treated as described in the text; the other, to the left, was radiographed just as it was removed from the chest.

upon the back of the chest, the following points should be noted: Firstly, are both apices of equal clearness? Secondly, what is the position of the heart? for I have noticed that in a case of pulmonary tuberculosis the heart is generally found in a much more vertical position than normal, although this depends somewhat on the side affected. The next point to look to is the working of the diaphragm. This is most important. Its importance has been insisted on by Dr. Francis Williams, of Boston, U.S.A. On getting the patient to take a deep breath, it will be seen that the diaphragm is rising and falling with much less freedom on the affected side than upon the sound side, and curiously enough this is frequently found in cases where the apex of the lung is apparently only affected. The chest, of course, should also be examined from the front after the back has been observed, because in this way we can sometimes pick up a shadow which might be missed by the posterior method of examination. Finally, the lateral oblique method of examination should never be neglected, because you will get some idea as to the amount of bronchial glandular enlargement by this method, which, of course, is secondary to pulmonary mischief. It is always as well, if time and opportunity permit, to examine the chest with the photographic plate, and also to examine it stereoscopically. By this means, I think, we can form a better idea as to the amount of tubercular mischief present than if we only examined the chest by the plate in the ordinary way. Of course, by bringing in the third dimennusion in space we get a better notion as to the extent of the pulmonary mischief, and here I would say that I think that this method brings us a little nearer to the time when it may be possible to treat by chemical rays pulmonary tubercle. It may be ultimately possible to accurately localise a tuberculous shadow so that it may be efficiently treated by chemical rays, so that, possibly, in the future we may say of tubercle in the lung "it comes as a shadow, so departs."

Now a word on the time of exposure in cases of pulmonary tuberculosis. In looking at the two following skiagrams, which are both of the same chest, taken with the same tube, the height of the tube above the chest wall in each case being the same, and taken at the same time—one after the other,—the first had one minute fifty seconds exposure, the second had five minutes exposure.

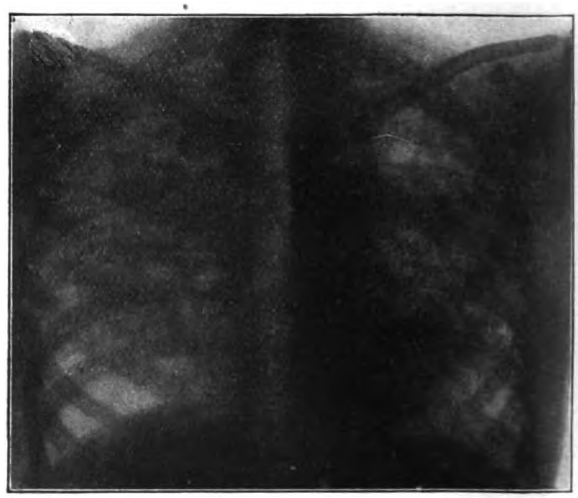


FIG. 12.—Chronic pulmonary tuberculosis. Exposure 1 minute 50 seconds.

Now on examining the skiagrams, it is at once evident that the tuberculous shadow is less in the one that has been exposed for the longer period of time. In the left lung this is especially

Digitized by Google

well shown; it obviously means that this length of exposure to the rays has penetrated some of the tuberculous shadow, and made it less dense. On comparing the skiagrams, we see that shadow corresponds to shadow in each lung, but the density is entirely different. This is, of course, an important point, because in an early case of pulmonary tuberculosis we may easily give too long an exposure, completely penetrating a tuberculous shadow, and showing a clear lung where really a shadow ought to be detected. On the other hand, we are in danger of giving too short an exposure, as the following case will well illustrate. A man with early pulmonary tubercle was exposed for fifty seconds, and on developing the plate nothing was seen to suggest that the patient had tubercle; while with a longer exposure of one minute twenty seconds the tuberculous shadow at the right apex was clearly and obviously discernable. But of the two there is less danger, perhaps, in giving a long exposure than in giving a short one, for, as I have just said, in the examples given above shadow corresponds to shadow, although the density of the one is less than the density of the other. I should now like to say a few words with reference to the employment of the screen and photographic plate in the diagnosis of tubercle. From my own experience I should say that one is certainly not more useful than the other, and both ought to be used. Frequently by making a screen examination we may detect a shadow at one or other apex, but nothing very definite; usually the same shadow comes out very definitely on the photographic plate. Therefore, if one is in any doubt at all as to the diagnosis with the screen, we should always go on and take a skiagram of the chest, because I am quite sure that shadows of tubercle are occasionally seen on the photographic plate which are altogether missed on the screen. This is probably due to the fact that the screens as now made are more coarse in texture, being covered with a coarse granular coating of platino-cyanide of potassium, while the gelatine film of the photographic plate has a much less granular texture, and shows up a shadow very much better than the screen.

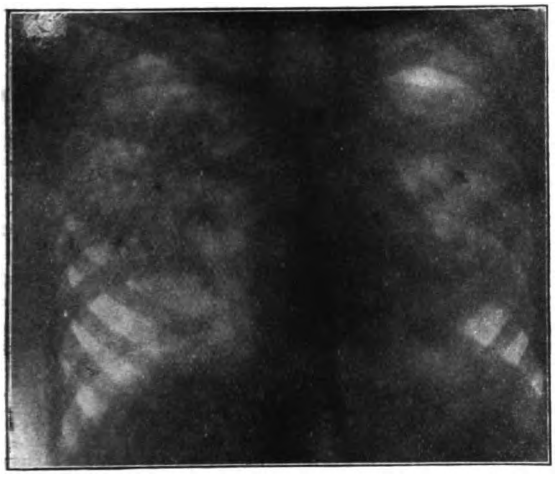


FIG. 13.—Chronic pulmonary tuberculosis of both lungs. Exposure 5 minutes.

I will now give clinically two early cases of pulmonary phthisis which came under observation in the out-patient room. A man æt. 22 came to the hospital complaining of cough and wasting of three months' duration. There was no family history of tubercle; there had never been any hæmoptysis. The sputum had been examined on many occasions for tubercle bacilli, but none were found. On examination the chest appeared normal, with the exception of a little prolongation of expiration at the apex of the right upper lobe; the skiagram, however, left no doubt as to the diagnosis (see Fig. 14). There was a distinct shadow occupying the right apex, and extending nearly to the level of the third rib. This patient was kept under observation, and finally the physical signs of pulmonary tuberculosis became quite distinct.

FIG. 14.—Pulmonary tuberculosis of right apex.

I will give one more case of so-called early pul-

Digitized by Google

monary phthisis, and with these I must be content. A woman æt. 25 came to the out-patient room complaining of cough, night sweats, and wasting of six months' duration. There was no family history of tubercle. The tubercle bacilli were not present in the sputum, although, however, it was only examined once. On examination there was dulness to percussion, bronchial breathing, and a few scattered crepitations at the apex of the right upper lobe; the left lung was apparently clear. The following skiagram is from this patient's

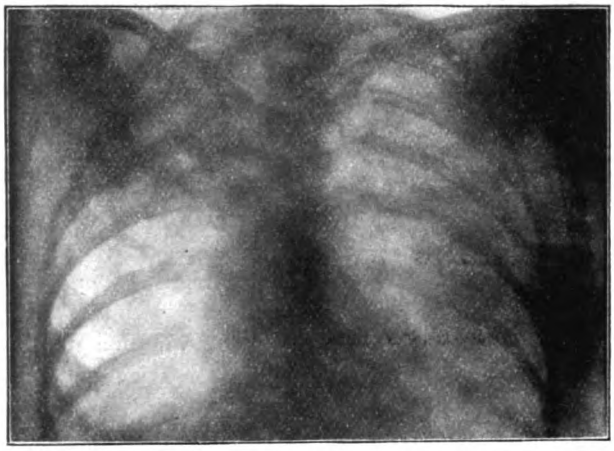


FIG. 15.—Pulmonary tuberculosis of right apex, showing flattening of affected side.

chest. We see at once that the contour of the two sides of the chest is markedly altered. There is distinct falling in of the upper ribs on the affected side, and there is a dense tubercular shadow extending from the apex downwards to the level of the third rib, or even a little below. We also see that there is beginning mischief on the left side, which the stethoscope gave no indication of. The next illustration I will give is one of advanced pulmonary phthisis. Clinically there was no doubt from the stethoscopic signs that this patient was in an advanced state of pulmonary tubercle of both lungs. There were no signs of cavitation. It will be seen from the skiagram that there is advanced disease of both lungs; the shadows being almost equally distributed on both sides from apex to base. I will now give a few examples of cavitation. That the Röntgen rays are capable of detecting quite accurately, and localising cavities of certain size within the lung, there can be no question; moreover, the rays frequently display the presence of a cavity in the lung where the stethoscope has given no indication

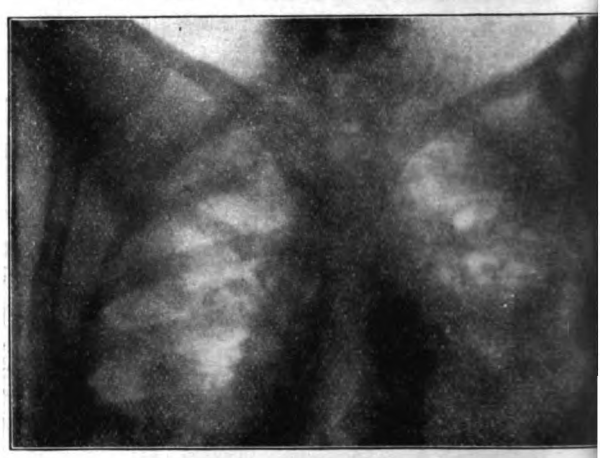


FIG. 16.—Advanced pulmonary tuberculosis of both lungs.

of its presence. The following skiagram is from the chest of a woman with advanced pulmonary phthisis. There is a large cavity at the right apex with general tuberculous infiltration of each lung. The cavity is denoted by a clear space surrounded by more or less dense shadow. Cavities are very

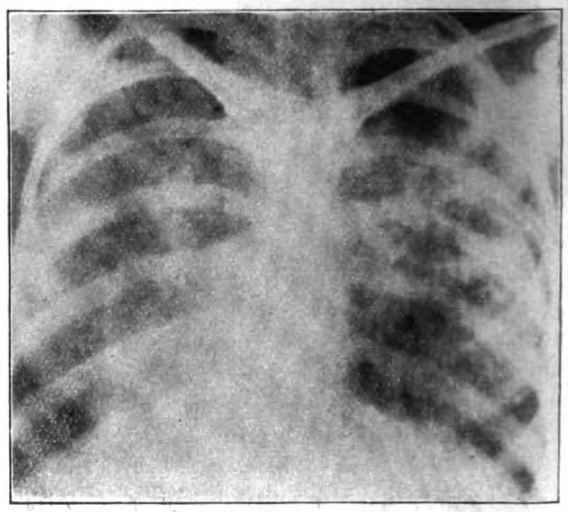


FIG. 17.—Advanced pulmonary tuberculosis of both lungs. There is a large cavity at the right apex.

frequently not as large when seen by the rays as auscultation would lead us to believe. It frequently happens in a case of pulmonary phthisis

Digitized by Google

that from auscultation one would diagnose a cavity extending perhaps from the apex downwards to the third or even the fourth rib, but on examining such a case with the X rays it is seen at once that the cavity is nothing like the size auscultation would lead us to expect.

A boy æt. 10 was admitted to hospital with advanced pulmonary disease, and on auscultation there were signs of a cavity on each side extending from the apex down to the level of the third rib. I was therefore surprised on examining the chest with the screen to find that the cavities were nothing like as large as one would have supposed from auscultation. There can be no question that solid lung surrounding a cavity will conduct the sounds formed in that cavity far beyond its limits.

The next illustration is a skiagram of cavities in

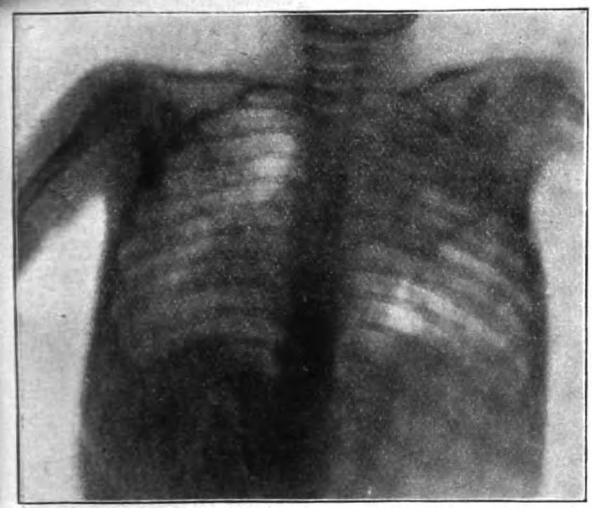


FIG. 18.—Chronic pulmonary tuberculosis, showing cavities at left apex and right base.

a child. This plate was kindly lent to me by my friend Mr. Payne, who is in charge of the X-ray department at the Sussex County Hospital. We see cavities at the left apex, and also at the right base. The case is an interesting one, because it was watched through to the post-mortem. This patient died with suffocative hæmoptysis due to a rupture of the aneurysmal dilatation of the pulmonary artery in the cavity situated at the base of the right lung. Perhaps of all the various cases of lung disease to which the Röntgen rays can be applied, that of fibroid or tubercular fibroid phthisis offers the most striking pictures. The following is an illustration of such a chest. A lad æt. 18 came to my out-patient room complaining of cough and dyspnœa of five years' duration. The skiagram shows a beautiful example of fibroid

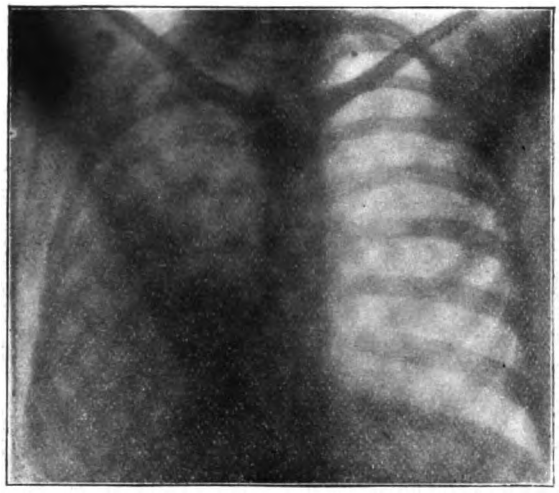


FIG. 19.—Fibroid phthisis of right side. Heart is seen to be displaced to the right side of chest; the clear left side stands out in marked contrast.

phthisis. The whole right side of the chest is in dense shadow. The heart is completely dragged over to the right side. The distended and clear left side of the chest stands out in marked contrast

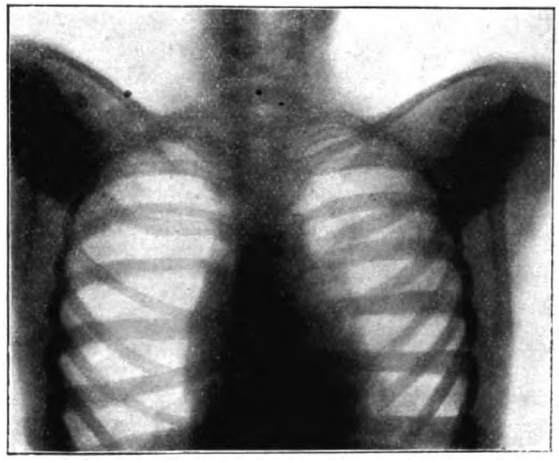


FIG. 20.—Pulmonary emphysema in a child.

with the affected side; the spine will also be noted to be somewhat curved to the affected side. There are many more cases of pulmonary phthisis I should liked to have recorded, but I must be con-

Digitized by Google

tent with the above. I will now pass on to the consideration of emphysema of the lungs. The next illustration is from the chest of emphysema in childhood. It shows very well the barrel-shaped chest, the horizontal ribs, the exceptionally clear pulmonary image on both sides, and the vertical position of the heart with the low position of the diaphragm. One word with regard to this vertical position of the heart in emphysema. This is the usual position of the organ in this disease, owing, no doubt, to the lowered position of the diaphragm. I think the usual explanation given of the obliteration of cardiac dulness in emphysema is erroneous. The explanation usually given in the text-books is that the cardiac dulness is abolished because we have emphysematous lung overlapping the heart, and thus separating it from the anterior wall of the chest; but if we look at the chest in the following skiagram of emphysema we shall see that the heart has an altogether vertical position, the apex-beat approximating to the epigastrium, and I think the cause of the absence of cardiac dulness in these cases is not due to emphysematous lung lying between the heart and the chest wall, but to the fact that there is no heart in the usual position to produce the dulness, the true apex-beat being in the epigastrium.

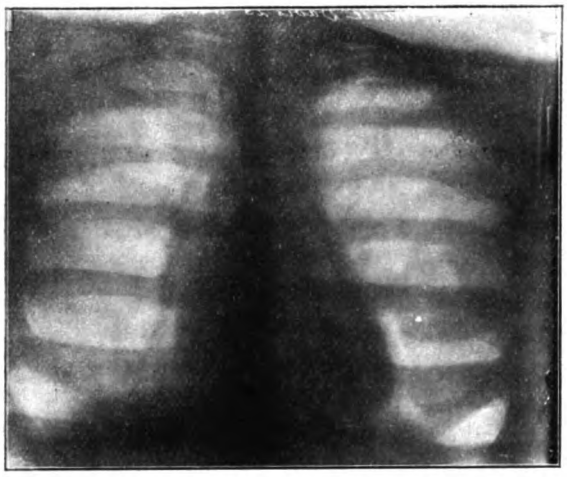


FIG. 21.—Pulmonary emphysema in an adult.

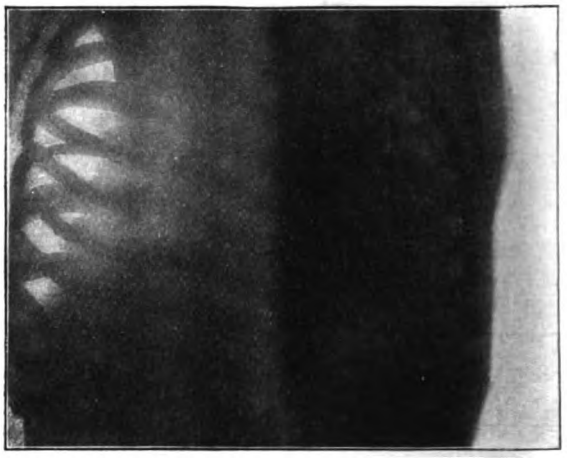


FIG. 22.—Pneumonia of the right lower lobe in a child.

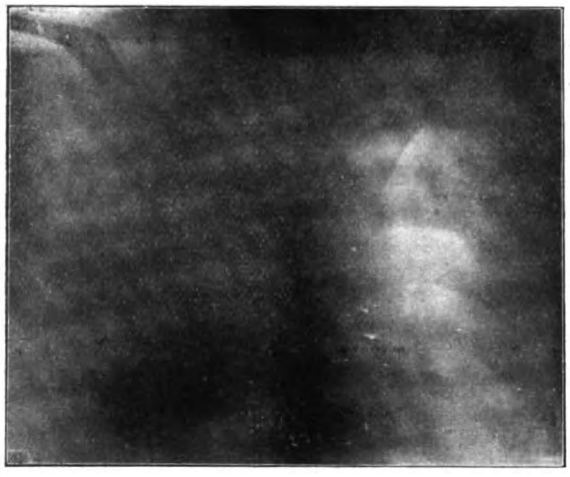


FIG. 23.—New growth of left lung.

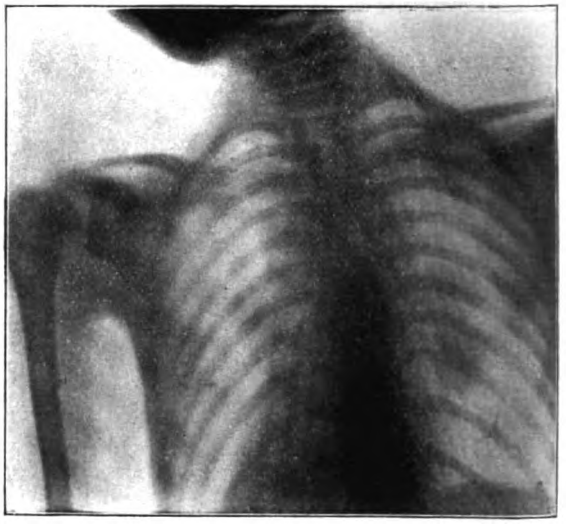


FIG. 24.—Enlarged bronchial glands in a child.

The next illustration is from a case of emphy-

Digitized by Google

sema in a woman thirty-two years of age. Again we see the same horizontal position of the ribs, the same clearness of the pulmonary image, the same low position of the diaphragm, and the very vertical position of the heart.

I now come to pneumonia. I have not had very many opportunities of taking skiagrams of cases of acute croupous pneumonia, the patient generally being too ill for any application of the rays to be made use of, so the following example of croupous pneumonia of the right lower lobe in a child must suffice. We see the whole of the right lower lobe in shadow. It may be asked, how is this to be distinguished from a pleural effusion, where we also see the lower portion of the chest in dense shadow? I think only in this way; with the screen the shadow of the fluid will change its position with change of position of the patient, whereas the shadow of the pneumonic lung remains unaltered. I will now pass on to the diagnosis of new growths in the lung. I have not had very many opportunities of taking a skiagram of a new growth in a lung, but some time ago I was asked by my colleague, Dr. Vincent Harris, to take a skiagram of a chest of a gentleman fifty-six years of age (Fig. 23). The whole of the left side of the chest, both back and front, was completely dull to percussion. There was tracheal breathing extending from the apex down to the border of the third or fourth ribs. There was a

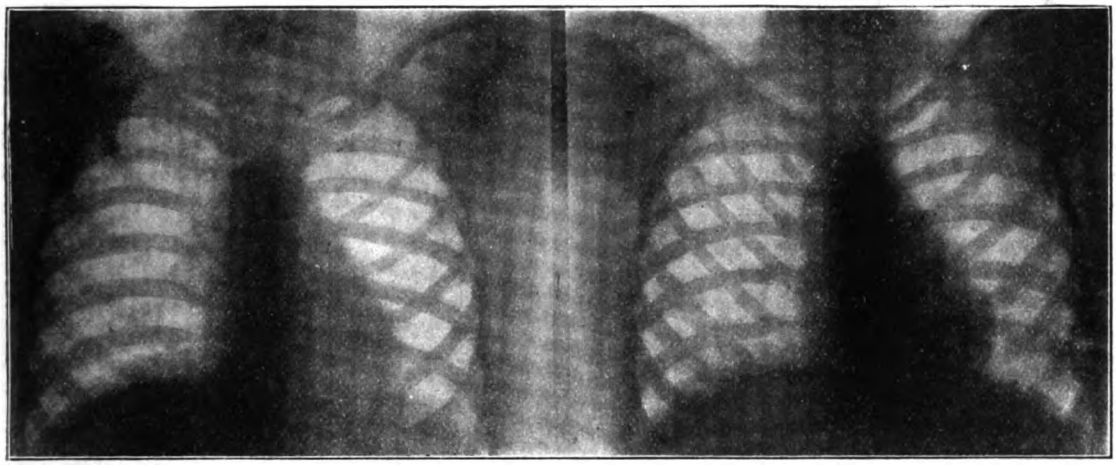


FIG. 25.—Steroscopic. Normal chest.

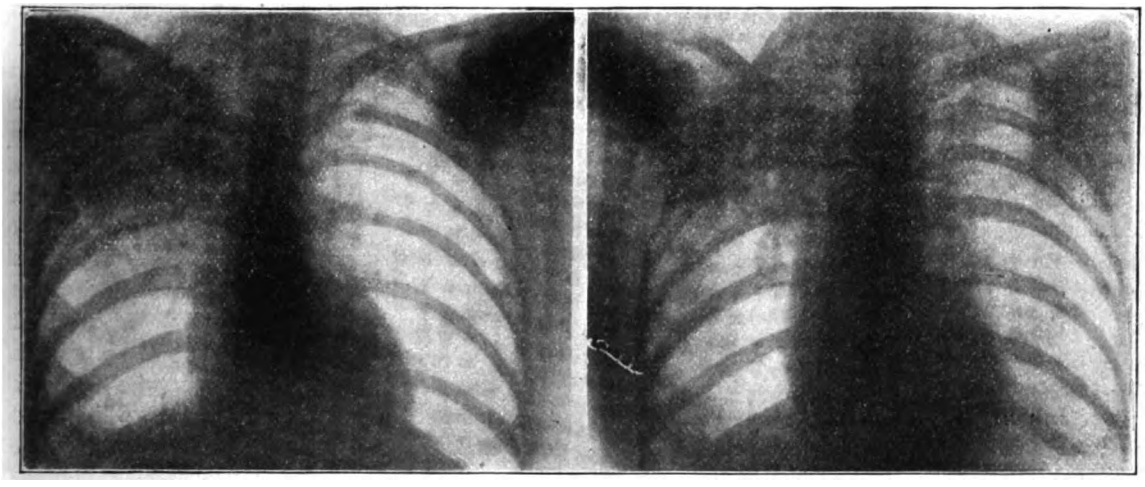


FIG. 26.—Stereoscopic. Right apical tuberculous consolidation.

Digitized by Google

considerable amount of dyspnœa. The expectoration when I saw the patient was mucous in character, but I was told that he had been expectorating pus, and there was some question as to whether the patient had a localised empyema. The skiagram shows a patchy ill-defined shadow all over the left side of the chest, and extending across the middle line slightly to the right side. The post-mortem in this case showed new growth in the left lung. Skiagrams of diseases of the anterior and posterior mediastinum, as I have before mentioned, can be obtained by the lateral oblique method of examining the chest, and occasionally in enlarged bronchial glands the retro-cardiac triangle may be completely filled up with shadow.

I will now give a case of enlargement of the bronchial glands. This skiagram was kindly lent to me by my friend Mr. Payne. It represents the chest of a child five years of age, who was admitted to the Sussex County Hospital. On an X-ray examination well-marked nodular shadows were found on each side of the spine in the position of the bronchial glands (see Fig. 24).

Finally, I give two skiagrams of a chest taken stereoscopically, first a normal chest, and secondly, one affected with tuberculous consolidation of the right apex. If these illustrations be placed in a Brewsters' or Wheatstone's stereoscope a stereoscopic effect will be obtained.

Living Animal Organisms in the Ear.—Francis R. Packard says that as a rule any animal within the ear may be killed and its remains removed by proper syringing with rather a strong solution of carbolic acid in warm water. In some instances merely holding a light in front of the external auditory meatus has served to attract the insect and cause it to make its way out, but the best way to produce relief is to flood the meatus with warm water or oil. C. H. Burnett recommends the use of chloroform through the meatus. Dr. Blake removed the maggots from a suppurating ear by holding a sponge saturated with ether at the external meatus. This caused all the maggots to wriggle out from the canal. Sometimes examination with a speculum has failed to reveal the presence of anything abnormal within the ear, and yet syringing will remove an insect.—*Penn. Med. Journ.*, April, 1901.

WITH MR. LOCKWOOD IN THE WARDS OF ST. BARTHOLOMEW'S HOSPITAL.

Cardinal Rules in Diagnosis.

I often used to hear Sir William Savory say that more mistakes in diagnosis arose from want of care than from want of knowledge. I suppose we should all be willing to take every possible care provided we knew in what that care consisted. Now it has often seemed to me that if you are careful in applying the simple rules of diagnosis which are always observed in this clinical class, you will be in a better position to avoid mistakes. Although we have gone over them so many times, I will ask you to repeat them once again, and I will endeavour to show you how they work out in practice. The first rule is to examine the whole patient; the second the whole of the diseased part or structure; the third to compare the opposite sides of the body; and the fourth is to examine the diseased part at rest.

Now let us begin with the first rule, which says that you should look at the whole patient. I need hardly point out how much may be inferred from what is seen in regard to the character, the social position, and the general health of the patient; sometimes a glance will tell us that whatever the local disease may be, we have to do with a serious illness. And here I may remark in passing that there are some who have had *much* to do with illness and disease, but who nevertheless do not seem to have acquired the faculty of knowing when a patient is seriously, perhaps fatally, ill. But let us see how the first rule may work in practice. I remember one day being asked to give an opinion upon the case of a young woman who had, as I was informed, a syphilitic eruption, which had obstinately refused to yield to treatment. The latter had, in truth, been vigorous enough, because I was informed that mercury had been given to the extent of causing salivation. The gums were ulcerated and retracted from the teeth; rather a calamity for a young woman, or, for that matter, for anyone else. Now in syphilis the stage of general eruption is always associated with anæmia. I have over and over again had the red cells counted and found them diminished by a quarter or one third of their number. Now a glance at

Digitized by Google

the whole of this young woman showed that she was not anæmic. Her cheeks were rosy, her eyes were bright, and her hair was lustrous. Further examination showed that other signs of syphilis were wanting, such as general lymphatic engorgement, ulceration of the fauces, and so on, and the rash was scaly and situated upon the extensor surfaces. So it became obvious that she was suffering from psoriasis, a disease which would not have benefited by a course of mercury, even although it had been pushed to salivation. I cannot help thinking that if the gentleman who showed me that case had been in the habit of applying the first rule in diagnosis, and had understood the significance of what he saw when he looked at the whole patient, he would not have fallen into such an unfortunate error.

We now come to the second rule, which says that you should examine the whole of the diseased limb or structure. Let us take the whole diseased limb first. A little while since I showed to this class a man who had a small ulcer upon the dorsum of the foot. He said that he had had an ulcer there for many years, but that latterly it had increased in size and become painful. The ulcer itself had a hard base which adhered to the tarsal bones. The class spent much time in looking at the ulcer, but failed to come to a correct conclusion. When the garments were removed and the whole limb examined, a mass of enlarged, hard, and adherent lymphatic glands were seen at the base of Scarpa's triangle. This, of course, threw a new light upon the nature of the ulcer. The man's statement was probably true. He had for years had a neglected ulcer upon the dorsum of the foot, which had at length become epitheliomatous, with secondary invasion of lymphatic glands. But this interpretation of the case did not occur to your minds until the second rule in diagnosis had been fulfilled. Think of your position had you been in charge of the case and overlooked the mass of disease in Scarpa's triangle. The case which we are about to examine also served to exemplify the second rule in diagnosis, which also says that you should examine the whole of the diseased structure. We have been looking at a tumour in the left side of the neck beneath the sterno-mastoid muscle. We have come to the conclusion that it consists of a number of enlarged lymphatic glands. We can feel enlarged lymphatic glands in the left subclavian triangle, and likewise in the right. Inasmuch as some of them are hard and others soft, we are inclined to think that they may be tuberculous glands. As you are aware, tubercle passes through the following stages :—First, the tubercle bacilli are deposited and excite inflammation. Next the inflammatory products degenerate and caseate, the caseous material softens, and a tuberculous abscess is the result. We think that the smaller glands that we feel may exemplify the earlier stage, that of inflammation ; the hard ones that of caseation, whilst the soft ones may contain pus ; but let us apply our rule and examine the whole of the diseased structure ; now we find that the lymphatic glands in the axilla are enlarged, likewise those in the groins. Where else is lymphatic tissue found ? The spleen contains a good deal. Let us see whether we can feel his spleen. We are unable to do so ; nevertheless we have ascertained enough to make us cautious what we infer about the glands of the neck, and to suggest that the case is in need of fuller investigation, especially as to the composition of his blood.

The third rule, which tells you to compare the two sides of the body, is constantly violated. How often in this class is wasting overlooked in cases of arthritis of the knee or hip because the opposite limb has not been examined. I remember once being shown a bony outgrowth on the spine of the scapula. It had been assumed to be an exostosis ; the surgeon in charge of the case ought to have been familiar with the trapezium tubercle, but even if he was not, a casual examination of the opposite side would have shown him that it possessed a similar outgrowth.

Now we come to the last rule, which says that you should examine the diseased structure in the condition of rest. We have just had an example of the meaning and importance of this rule. Some of the class were asked to say whether the tumour of the neck adhered to the deeper structures, such, for instance, as the carotid sheath, the scalene muscles, or the vertebral column. Finding it immobile, they assumed that it had formed these deep connections, and they further seemed inclined to think that the disease within the glands, whether inflammatory or malignant, had passed beyond their capsules and caused them to adhere. This assumption might, of course, have been correct, but they failed to observe that they were

Digitized by Google

violating the rule in diagnosis; each time they endeavoured to move the mass they allowed the man to contract the platysma, the sterno-mastoid, the trapezius, and indeed, all the muscles of the left side of the neck, and this suffices to render the mass immobile. Now ask him to relax the muscles, and observe that the tumour can be moved with considerable ease.

Of course to-day we have only touched the fringe of a large subject. Some cases are most difficult to examine. A thorough examination may be impracticable, unless the patient is kept for some time in bed under immediate observation. It may be necessary to make an endoscopic examination of the urinary bladder, to examine all the special organs with special instruments, to test the electrical reaction of muscles, to take radiographs, to examine under an anæsthetic, and perhaps to perform an exploratory operation. But the latter should only be undertaken when all the other means of diagnosis have been exhausted. I have said but little to-day concerning the *methods* of diagnosis, but you will observe that, as usual, we have endeavoured to obey that great teacher, Sir George Humphry, who said, "eyes first and much, hands next and little, tongue not at all."

Retroflexion and Retroversion of the Gravid Uterus.—H. Keitler insists that backward displacement of the pregnant uterus may occur from a change in position of a previously anteflexed organ as well as in one retroflexed or retroverted before gestation. Retroflexion may result in retroversion, and *vice versâ*. Differentiation of these positions is essential, as spontaneous reduction of a retroverted uterus is not to be expected, and active treatment is more imperative than for retroflexion. Cases of retrodeviation with a very short vaginal portion of the cervix, or only an external os with the os directed forward and upward, possess the practical significance of a retroversion. The resulting retention of urine must not be treated by sudden complete removal of the contents of the bladder, whether these are affected or not. The so-called hæmorrhage from creation of a vacuum is always a sign of beginning necrosis of the bladder.—*American Journ. of Obstet.*, June, 1901.

THE PRACTICE OF BLOOD-LETTING.

By J. F. BRISCOE, M.R.C.S.

(*Continued from page* 96.)

I proceed now to the consideration of venesection in apoplexy and head injuries.

When there is a collection of fluid in any organ or tissue of the body, the first thing that appeals to us is to effect the relief of the tension of this fluid; so that in cerebral hæmorrhage the object of the physician is to retard the circulation and stay further hæmorrhage. The typical apoplectic is plethoric in habit; the appetite is generally large; and the vascular column, always more or less distended, may give way at any weak spot in its walls. The skin of the body is often surcharged with fat, and the organs generally more or less in a state of fulness. It is by the frequent action of the kidneys and bowels that the plethoric subject is enabled to expel the excess of fluids from the tissues. Should a person of this description neglect to take sufficient exercise or indulge the appetite too freely, he or she not unfrequently runs the risk of cerebral hæmorrhage, or another incidental accident of congestion. From this, then, it is obvious, that there is no better class of patient suited for the practice of blood-letting than the plethoric. Sir Dyce Duckworth informs me that in his practice of blood-letting he has not been struck by any decided advantage produced by venesection in case of cerebral hæmorrhage in the majority of his cases. He says, "I usually bleed at once in plethoric subjects so attacked I take it the question of recovery really turns on the amount of blood poured out into the brain. It may be, however, that a bleeding may help to moderate and arrest an ingravescent hæmorrhage." However, it is impossible to lay down definite ruling that will always guide us even in cases of cerebral hæmorrhage.

At the meeting of the Royal Medical and Chirurgical Society in 1891 the therapeutic value of venesection was the subject for debate. The paper was communicated by Dr. Pye-Smith, being a record of all his cases of venesection, extending over several years, and I believe the first, except Sir Samuel Wilks, to publish nearly fifty cases. The opinion expressed by the majority at this meeting

Digitized by Google

was that a valuable weapon, if rightly used, had been thrown away. In his reply Dr. Pye-Smith remarked "that everyone would be sorry if its revival were to lead to abuse." More particularly in cerebral hæmorrhage is this abuse likely to occur, for we are just as much at sea now as were our forefathers with regard to any rapid method of locating a cerebral hæmorrhage. To bleed everyone for hæmorrhage into the brain is ridiculous in the extreme; it would only add abuse to injury, and again history would repeat itself. For instance, in ventricular bleeding, what is the prospect of recovery? While in cerebral congestion, with the premonitory signs of rupture, blood-letting may save the life of an elderly subject. Nay, it is possible to save a life when a less vital part of the brain than the ventricles have been subjected to pressure of blood from the oozing of a diseased vessel. Moreover, there are cases of capillary leakage, either local or general, in which the physiological action of blood-letting is second to no other remedy as an absorbent. Are we not then justified in asserting that in the many cases recorded in the medical journals of successful venesection in cerebral hæmorrhage that we have a weapon in blood-letting which should not be despised? But if the practice of blood-letting is again seeing the light of day, let us one and all remember the errors and abuses of routine practice. The operation of blood-letting in practice requires the skill of a shrewd practitioner, and unless he has in his mind's eye, for instance, the picture of an impending case of cerebral hæmorrhage he is working in the dark. It is important for him to have at his fingers' ends the details of those cases of cerebral hæmorrhage which are slowly ingravescent, as by judicious skill he may avert a fatal issue. It is when reaction appears in these cases that blood-letting will give the best results. The desire is to lower the pressure in the cerebral vessels and prevent further effusion of blood. Just as we slacken the current in popliteal and other aneurysms by slowing the circulation, so do we relieve cerebral hæmorrhage by blood-letting. Again, in aneurysms where pressure cannot be applied, iodide of potash in conjunction with potash is worth a trial. Dr. Pye-Smith advocates venesection in the pain and distress of aneurysm, and he states that he learnt this from the late Professor Hughes-Bennett of Edinburgh. The point of Dr. Pye-Smith's teaching is, "bleed for pathological indications—cyanosis, distended right heart, eclampsia, pain, etc.—not to cure diseases." In the London Hospital Museum there is a specimen of an aneurysm showing perfect cure brought about by repeated bleedings. Mr. Hutchinson, senr., 'Lancet,' January, 1891, says "that surgeons were constantly seeing patients who had lost much blood, and who apparently were none the worse for it." However, he would prefer to venesection what he calls a simpler measure in head and chest injury, namely, placing the feet up to the knees in a bath of very hot water for a considerable period.

But this line of treatment in head and chest injuries must lead to so much strain and inconvenience to both patient and physician that I beg most respectfully to question whether considerable periods of the semi-upright position in a hot bath is the best management of chest and head injuries. The late Mr. Hulke quotes a case of head injury in which the abstraction of one pint of blood entirely relieved a condition of cyanosis and convulsion. Moreover, Mr. Pollock recommends blood-letting where ribs are fractured and the lung is perforated, there being hæmoptysis and a condition of great distress. Dr. Broadbent, now Sir William Broadbent, in the discussion on Dr. Pye-Smith's paper, said "he had seen life saved in mitral stenosis and mitral regurgitation by venesection, but he was brought up in an anti-venesection school." However, Sir W. Broadbent, in the discussion at the Medical Society, November 26th, 1900, "On the Treatment of Thoracic Aneurysm," says, "but free bleeding afforded the greatest relief to breathlessness and pain," and he referred to a case in which the patient had lived many years afterwards. The same authority failed to get blood from the vein in a case of ingravescent apoplexy owing to the excessive peripheral spasm, and also in another patient with uræmic and acute distension of the right heart the blood would not flow. The failure to obtain blood after a vein is opened is not an infrequent occurrence, but patient waiting, provided the vein is large enough and has been opened *secundum artem*, may be rewarded with success. Dr. Talfourd Jones, of Eastbourne, "On Nitrite of Amyl," 'British Medical Journal,' 1871, has been successful in encouraging the flow of blood in wet cupping after the incisions have been made by the blades of the scarificator, by

Digitized by Google

relaxing the vascular system. This he did by an inhalation of nitrate of amyl, although previous to the administration the blood did not flow into the cupping glass. This method would seem to me equally felicitous should blood fail to flow when a vein is opened, although I have never adopted it. I have previously alluded to spasm in the form of convulsion, which is but vaguely understood physiologically or pathologically. The irritability may be due to something generated abnormally within an organ or a tissue, or it may be due to a foreign enemy circulating in the vascular system. Dr. Robert Lee, in the same debate on "The Therapeutical Value of Venesection," hoped that the whole subject would be worked out from the beginning, and he felt that in the arrest of local inflammation bleeding would again be resorted to with advantage. But until the student is encouraged in the practice of blood-letting, the practice will still remain relatively as "one of the lost arts."

There is a tendency to overvalue blood in these days, although large amounts escape, as in enterica, etc., without injurious consequences. But when the patient is *in extremis* we grudge its loss because we think it lowering; because we are ignorant of the use and value of blood-letting; because we are inexperienced in the art; and, finally, because we are carried away by every new doctrine, and influenced by some whose practice of blood letting is decidedly limited.

There is also another hindrance to the successful practice of blood-letting, and that is a large majority of practitioners are content, as Dr. Berry says, "to leave the lancet in the pocket of the consultant, who then might get the blame, if any were incurred for its use." The public are just as well aware of the disuse of blood-letting as we are ourselves. That the intelligence department of the profession has shown evidence of weakness at various epochs of the history of medicine is not to be denied; and let us hope in the new century there will be less of this absentmindedness in medicine and surgery.

It would be unnecessary waste of time if I were to enumerate a list of cases from medical journals of the beneficial effects of venesection in head and chest injuries, but the practical text-book of Mr. Bryant is worthy of the notice of all practitioners. He says, page 41, vol. ii, of his work, 4th edition, "Bleeding is now rarely performed, and at Guy's Hospital I believe it is rarer than any capital operation. In the case of lacerated lung, however, when urgent dyspnœa makes its appearance and the powers of the patient do not forbid it, I know of nothing which gives greater relief to the patient or greater satisfaction to the practitioner. Bleed with no sparing hand, letting it flow freely in a full stream, and as it flows, the symptoms will gradually disappear. When relief has been obtained, arrest the flow immediately, as syncope can only do harm. The aim should be to make an impression through the systemic circulation upon the pulmonary. The patient should be carefully watched, and the operation repeated if the symptoms return, and, if necessary, even for a third time." Mr. Bryant quotes a very good instance of a case, and the patient did well although six ribs were broken and a clavicle was dislocated. Again, in the treatment of concussion and compression of the brain, and of fractures of the skull, Mr. Bryant is a strong advocate of venesection. "It is," he says, "a valuable remedy when the symptoms of excessive reaction are persistent, and the commencement of traumatic encephalitis is suspected." Before I close the opinions of this practical teacher I should like to say that while some writers have nothing to say against the practice of venesection, they remark, however, that having little or no experience of it they are unable to recommend it. Dr. Shaw Little, of Cilfyndd, Pontypridd, Wales, whose appendix in "The Causes of Death in Colliery Explosions," by Dr. John Haldane, is so instructive, writes to me as follows: "Looking backwards in my own practice, apoplectic conditions have been exceedingly rare. But in connection with the terrible explosion that happened here six or seven years ago where nearly three hundred were killed, of those who were brought up alive suffering from the effects of after-damp, there were some whose condition appeared as truly apoplectic as if it had been produced by a hæmorrhage into the brain. I did what I could for all, including administration of oxygen, but blood-letting was not thought of. Dr. Haldane, who was much interested in experiments with CO (which gas was not found here in blood of living men or dead horses) saw several of the sufferers while still alive, and he did not suggest it either."

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 454. WEDNESDAY, JULY 10, 1901. Vol. XVIII. No. 12.

CONTENTS.

PAGE

*A Demonstration of Cases at the Medical Graduates' College and Polyclinic. By P. J. FREYER, M.D., M.Ch. 177

*Present Day Skiagraphy. WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL. Demonstration III 184

*Four Clinical Lectures on some Affections of the Kidney. By A. H. TUBBY, M.S., F.R.C.S. Lecture IV 187

*Remarks on the Summer Diarrhœa of Infants. By J. D. WINDLE, M.D. ... 191

Heroin. E. HAWKINS, M.A., M.D. ... 192

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A DEMONSTRATION OF CASES AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

May 8th, 1901.

By P. J. FREYER, M.D., M.Ch.,
Surgeon to St. Peter's Hospital.

TOTAL EXTIRPATION OF THE PROSTATE.

GENTLEMEN,—Before having in the patients I will refer to a case which I described to you at the last lecture, a patient from whom I removed the prostate completely, and showed you the three adenomatous tumours which were removed. Captain Pinch will show you microscopical sections of the tumour, from which you will see that they were true adenomata of the prostate. Since I last met you here I have had the good fortune to have come across an exactly similar case, which I will briefly refer to. I send you round the tumours which were removed, and a drawing showing the grouping of these tumours as they lay in the prostate. It was a case which happened in my private practice, the patient being a gentleman aged sixty-seven. Three and a half years ago he had the symptoms of enlarged prostate, viz. increased frequency of micturition, difficulty in starting the stream, diminution in the strength of the stream, straining, dribbling of water immediately after micturition, and intermittency of the flow. His private practitioner advised him to pass a catheter every night, which he continued to do for some time, in fact, until January of last year, when he came under my observation. I found that the symptoms had continued, but were much aggravated. On passing a catheter I found that there were no less than nine and a half ounces of residual urine. In addition to the other symptoms there was great pain in the hypogastrium, and he suffered periodically from severe attacks of gout. On examination by the rectum I found both lobes very much enlarged, rounded, slightly nodular, and soft, and I came to the conclusion that it was an ordinary adenomatous enlargement

Digitized by Google

of the prostate. As I have said, he had nine and a half ounces of residual urine, and I therefore advised him to pass a catheter three or four times a day. I examined him from time to time, and on the 19th March last he had complete loss of power over his bladder, so that he could only pass his urine by the aid of the catheter. He used his catheter now five or six times a day; his urine was thick and had a fishy, offensive odour, and was found under the microscope to contain epithelial cells and leucocytes. I examined the patient with the cystoscope, and found the appearance which I show you in the picture which I have sent round. There were two large tumours, each the size of a chestnut, representing the lateral lobes of the prostate, and projecting into the bladder, inwards and upwards; and between these two there was a deep vertical slit. On the base of the bladder near its neck there was a small tumour the size of a goosebery; it was very nodulated, resembling five or six split peas covered by mucous membrane. I opened the bladder supra-pubically nine or ten days afterwards, and found the conditions exactly the same as I had seen with the cystoscope. I introduced a pair of forceps, grasped first the left tumour, or the most prominent lobe, by the mucous membrane; then introduced a pair of scissors and snipped the mucous membrane over the tumour. Then I withdrew the instruments, passed the point of my finger through the opening made in the mucous membrane, and by means of the fore-finger enucleated one of these large tumours which you see in the bottle. There was no difficulty in getting this out from its bed; it was simply a question of slightly tearing through the fibrous tissue by which it was connected with the surrounding structures. Having done this, I attacked the right lobe and removed it exactly in the same way; and, introducing my finger through one of the slits in the mucous membrane, I hooked it round the middle lobe and removed it. In removing the lateral tumours I felt that my finger passed forward to the triangular ligament. My assistant, with his finger in the rectum, was able to make the tumours more prominent in the bladder and keep them steady; in fact, there was thus counter-pressure to my finger inside, so that the tumours were much more easily removed in this way. After removing these three tumours, I passed my finger into the rectum, and, with one finger in the rectum and the other in the bladder, felt that there was no prostatic tissue whatever left, the prostate being wholly removed. The enlarged prostate in this case, as indeed in a large number of cases, consisted of these encapsuled tumours. They were practically separate, but agglutinated together by fibrous tissue. To cut a long story short, I may say that the patient made a thorough recovery, and that he left the surgical home a week ago; that he can now pass his water without a catheter, that he can retain his urine four or five hours in the daytime, and that he can go the whole night without the necessity of getting up to pass it. I passed the catheter just before he left the Surgical Home, and ascertained that there was not half a teaspoonful of residual urine left behind. Therefore the case may be described as a thoroughly successful one. I have seen no cases like these in literature, in which the prostate was completely removed and in which a thoroughly successful result ensued.

Captain Pinch said the growth was a perfectly pure adenoma. The difference between that and prostatic tissue was that the unstriped muscular fibres had been replaced by fibrous tissue. The acini had no ducts, which was a feature common to all adenomata. These adenomata must have started and gradually grown, and by pressure the whole of the gland substance proper had been atrophied, so that when Mr. Freyer incised the mucus membrane he removed all that was left. The adenomata destroyed all the tissue of the prostate gland proper.

Mr. Freyer (continuing) said there was no bleeding connected with the operation; no vessel had to be tied during it; the mucous membrane was left to form a canal, and no doubt the parts filled up as they contracted, much in the same way, perhaps, as the womb contracts after parturition. You can only diagnose cases which are suitable for this operation by means of the cystoscope; that is, when you have distinctly prominent tumours projecting into the bladder. I do not mean to convey the impression that this operation is suitable for every case; in fact, it is only suitable for very rare cases indeed. I dealt with that subject in November last, when I lectured on the prostate in this building. I believe prostatic enlargements have not as yet been properly accounted for; but it is certain that the enlargement sets in as the

Digitized by Google

sexual powers decline. The prostate is undoubtedly a sexual organ. The modern theory to account for tumours is that they are due to "rests" and "vestiges," and I think the prostate may be regarded as a vestige when its physiological function ceases.

Suspected Tuberculous Kidney.

Next I show you this man, æt. twenty-two, brought for consultation by Dr. Gabe. He is unmarried. He complains of a continuous aching pain on the right side "in the small of the back," and the pain has been confined to that spot for seven or eight months. He describes it as a dull, heavy pain, and severe in degree. It does not shoot downwards from its apparent origin, but upwards to the back of the shoulder. The pain is constant, but it is worse sometimes than at others, namely at night rather than in the daytime. He is a painter. Stooping, we are told, makes the pain worse. He has experienced no trouble in passing his water, and urinates regularly about six times a day. He has not had to get out of bed to pass it. The water has not been passed more frequently since the pain came on. He says the urine sometimes looks like chalk, but it has never been red, though sometimes it has been of an orange colour. He has not seen any blood in it. The water is not passed more frequently when the paroxysms of pain come on. He has not felt any pain in the penis or bladder. He has had epileptic fits, and was invalided out of the Army on that account. His grandfather died of consumption, but he was an old man. There are four brothers and one sister in the patient's family, and they are all alive and in good health. Well, this is a case in which the symptoms are rather obscure. The only prominent symptom which he complains of is pain in the right loin, in the region of the kidney, and this pain has been continuous for seven months, gradually increasing in intensity, and there is not a very marked difference in the character of the pain from time to time. It appears not to be accompanied by any irritation of the urinary passages; there is no pain in the penis and no increased frequency of micturition. The only thing which he notices in connection with his water is that it is thick. Of course we have to ascertain by examination what the nature of this thickness is. It may be due to urates only. In cold weather, in people who are practically in good health urates are thrown down. Patients will come to you in private practice frightened about the appearance of their urine, and you will find there is nothing the matter with the urine except the presence of urates. That may possibly be the case here. But, on the other hand, it is possible that his urine may contain pus. There is no marked family history of tuberculosis. The case does not present the symptoms of stone in the kidney, because in that disorder you have the symptoms of severe renal colic; the pain is extremely distressing at times, accompanied by nausea and vomiting, the patient rolling about in the intensity of his agony. That is not the case here. In stone in the kidney there is, as a rule, hæmaturia; and you also have hæmaturia in tubercular kidney. This patient has no hæmaturia. In stone in the kidney there is increased frequency of micturition, and other symptoms to which we will refer presently. The next thing we have to do is to ascertain whether there is a tumour in his loin, and if so, what the nature of it is. When you have a suspicion that a kidney is diseased, it is better to commence by examining the other kidney, so as to draw a comparison between the two. On the left side I cannot feel the kidney, which is as it should be. You will notice that he has superficial pain on palpation on the right side. When I place my hand on the right loin and push the fingers in between the twelfth rib and the crest of the ilium there is distinct tenderness and pain. That is a symptom which you generally find in stone in the kidney. But I can feel no tumour here either; indeed, I cannot feel the kidney on either side. I cannot find any definite symptom of tubercular disease on this side, though it is possible that this does exist. You must remember that when tubercle attacks the kidney or kidneys, it attacks them in two forms. One is the miliary form, and the other is the cheesy form. In the case of miliary tubercle of the kidney, which generally occurs in young people, there is practically no enlargement of the organ, and there are scarcely any urinary symptoms attached to miliary tuberculosis of the kidney. On the other hand, when you have cheesy tuberculosis, you find enlargement of the kidney. It is quite impossible to establish a

Digitized by Google

diagnosis in this case until we have had a report upon the urine. If the patient has got pus in his urine, and particularly if tubercle bacilli be found, I would undoubtedly state that he has tuberculosis of the right kidney. Captain Pinch now tells us that his urine is 1018 specific gravity, that it contains some albumin and a good deal of pus, and that the tubercle bacillus has not yet been demonstrated. Of course, you know it often requires repeated examinations to detect the tubercle bacillus, so that a negative in this respect counts for very little. Considering the family history of the man—I am told the mother is in a very delicate state of health and that the grandfather died of phthisis—and that the patient looks as if he may be more or less phthisical, I think it is very likely that he is suffering from tuberculosis of the right kidney, particularly as there is pus in the urine. I will keep him under observation, and have his urine examined several times to see if the tubercle bacilli can be found. In order to complete the diagnosis it will be advisable to introduce the cystoscope into the bladder and ascertain where the pus comes from. First, I shall have a look at the bladder to see if it is tuberculous. Probably it is not, as there are no symptoms of it. There is no excess of vesical epithelium in the deposits. So we have to suspend our judgment in the diagnosis of this case until we have a further report upon the urine.

Primary Tuberculosis of the Bladder.

The next patient is a man æt. 27, who has been suffering for three years from hæmaturia. He experiences pain at the end of the penis during micturition, and the pain passes along through the organ down to the perineum. He has to pass his water every half hour by day, and six or seven times at night. The symptoms are increased on his taking exercise. His urine stops, and then goes on again several times. His father died of consumption; but the patient has five brothers and two sisters, and they are all alive and well. The hæmaturia is worst in the morning when he gets out of bed. He says that when he passes his water he pumps out a little at a time,—that is to say, the stream is intermittent. Sometimes the pain is so severe, that he has "to hold himself," and, in fact, there is pain along the penis, in the hypogastric region, and all about the region of the bladder. There is pain in the perinæum also. He has never been without pain in this way for eighteen months. He has been passing blood in his urine on and off for three years. During the first eighteen months he passed blood without pain. He says there have been distinct clots in the urine sometimes. I have examined this man's kidneys, but could not find any enlargement whatever. The symptoms might be those of stone in the bladder or tubercle. The symptoms are not, however, at all characteristic of stone in the bladder. The pain in connection with stone in the bladder is, as a rule, confined to the tip of the penis, and occurs just towards the end of micturition and immediately afterwards. The patient suffers pain during the whole time he is passing water, as well as immediately afterwards. I sounded this patient lest there might be a stone, but found there was none. I then admitted him into St. Peter's Hospital, and on Wednesday last I made a cystoscopic examination of the bladder, and the only appearances I found were that there were two small ulcerations, one inch outside and behind the right ureteral opening. These ulcers were saucer-shaped, with the edges slightly raised; and the mucous membrane all about them was highly congested for a distance of about a quarter of an inch. On the surface of each of these ulcers was a soft phosphatic crust. The rest of the bladder was perfectly healthy, and the mucous membrane in the vicinity of the two ureteral orifices was normal. I cannot tell you definitely whether all the blood which the man states he has passed was due to these two ulcers; you must remember that patients exaggerate when they see the urine coloured; if it is coloured they think the quantity of blood in it must have been considerable, whereas very little blood will produce distinct colouring. He is losing flesh, and has distinctly stated that he has passed clots of blood, and it is possible that the clots issue from these small ulcers. I do not think there has been any blood in the urine since he came under my observation. Even after the examination with the cystoscope he passed his water quite clear. We have not made an examination of the urine to ascertain if there are tubercle bacilli, but from the family history I have no doubt that this is a case of tuberculosis of the bladder. If there be no disease of the

Digitized by Google

kidney, and I do not anticipate there is such disease, as he has no tenderness of the kidneys, no enlargement, and no pain on examination. I should say it is a case of primary tuberculosis of the bladder. Now, primary tuberculosis of the bladder is a very rare disease indeed. I may say I have also examined this man's genital organs—testicles, epididymes, prostate, and vesiculæ seminales,—but there is no sign of tubercle in any of those organs. So we are reduced to believing that the symptoms are entirely due to those two small ulcerations of the bladder, which are tuberculous. So far as one can see, this is a distinct case of primary tuberculosis of the bladder. There is no evidence of tubercle in the lungs or elsewhere.

I had better run over with you some of the symptoms of tuberculosis of the urinary organs in general, and particularly with reference to that in the bladder. When tubercle attacks the bladder, you generally find the evidences of tubercle immediately around one or both of the ureteral openings into the bladder, or on the trigone. When the disease is primary, it may occur in any part of the bladder. When the disease extends from the lower urinary and genital organs, that is, when it extends from the urethra or from the prostate, testicles, or vesiculæ seminalis, it is found in the trigone. And when you have the disease attacking the bladder, as a descending tuberculosis, when it creeps gradually down from the kidneys, you have the appearances about the ureteral openings. Tuberculosis of the bladder is generally found in two particular parts—at the ureteral openings and on the trigone. Now, the appearance of tubercle of the bladder when it first commences is that of small greyish raised nodules, and these nodules after a time form cheesy matter, and then soften and suppurate, and an ulcer is formed. You may have a number of these isolated ulcers scattered about the ureteral openings, and about the trigone. These small ulcers, in time, have a tendency to coalesce, and thus irregular ulcers are formed. These ulcers are always depressed below the surface of the mucous membrane, as if a patch were punched out of the mucous membrane, the edges being slightly raised. The zone immediately in contact with the ulcer is pale and anæmic. Immediately outside this the mucous membrane is highly congested, like maroon velvet, and in that zone, which extends for about a quarter or half an inch outside the ulcer, you will frequently find fresh tubercles, which, of course, in time break down, and form fresh ulcers. In the early stage of the disease, when the urine is acid, there are no deposits on these ulcers; but, unfortunately, in the course of time cystitis has a tendency to occur, the bladder gets infected with pus, and then the urine becomes alkaline, and you find these ulcers covered over with phosphatic deposit. You will also find that the ulcers are covered over with muco-pus. If you look at the ulcers with the cystoscope, you find that they are covered by these matters I have mentioned; and you also see that there are shreds hanging out from them into the bladder. Another thing in connection with them is that the parts around the ulcers are extremely raised, like the points of one's fingers, very like sessile fibro-papillomata. Primary tuberculosis in the bladder is, as I have said, a very rare disease. It is generally secondary to tuberculosis in some other part, generally the kidney or genital organs. When the disease extends to the bladder from the kidney, it may occur in one of three or four ways. In the first place, it may occur from infection through the blood-stream, or it may occur through the lymphatic stream; but the general way in which it is conveyed is by extension along the mucous membrane of the ureter. The kidney gets gradually involved, and then the ureter, and the disease gradually extends down into the bladder. You find the ureteral openings into the bladder congested and nodular, just as I have already described to you. In the same way, when the testicle, epididymis, prostate, or vesiculæ seminales are infected, the disease creeps along the mucous membrane of the prostate and into the bladder, attacking the neck of the bladder and trigone. Another way in which the bladder may be infected is by direct ulceration of the tubercle from the prostate. These ulcerations may break down and burst directly into the bladder, and in this way the bladder may become infected.

I have said that there are two kinds of urinary tubercle, the miliary and the cheesy. Urinary tubercle, as a rule, is found in patients of from fifteen to forty-five years of age, but I have frequently seen tubercle in children of eight or ten years of age; and the younger the patient is

Digitized by Google

the more likely you are to have miliary tubercle. That form, as a rule, attacks both kidneys; it is very rare for it to be confined to one kidney. You may therefore take it generally that in the case of operation for tubercle of the kidney the operation is much more successful in persons as they advance in life than in younger people, for the simple reason that the other kidney is much more frequently attacked in young people than in adults.

Now, what are the symptoms of tubercle of the bladder? You have already heard that this man has increased frequency of micturition. An important distinction between the frequency occurring in tuberculosis and that occurring in stone in the bladder is that in tuberculosis you have increased frequency as much at night as in the daytime, whereas in stone in the bladder the increased frequency is chiefly noticeable when the patient is going about in the daytime. A person with stone generally finds there is no necessity for him to get up at night to pass his water. The next symptom is pain in connection with the act of micturition. This patient tells us he has pain along the whole of the penis and hypogastrium and in the perinæum. The pain in connection with stone in the bladder is almost invariably at the end of the penis, and it also occurs towards the end of micturition and immediately afterwards, whereas in this case the pain occurs during micturition as well as immediately after. There is an undefined character about the pain in this case which does not occur in stone in the bladder. There is general pain all over the region of the bladder and genital organs. Next we have hæmaturia. In hæmaturia in connection with tuberculosis you do not find, as a rule, that the bleeding is very profuse, and I do not think it has been in this case. It is a fortnight since he came under my observation, and during that time there has been no bleeding. Still, undoubtedly he has passed blood, and the ulcers which I have described indicate that he has passed it. In stone in the bladder, also, you have hæmaturia, and that also is not of a very severe character, whereas in tumour of the bladder the hæmorrhage is always very profuse. In this case he had sudden stoppage of the water. He has not at all stoppage like that which occurs in stone in the bladder. In the latter case the stone blocks the passage, and the patient is unable to pass his water for some time; in fact, he generally lies down, and after a time the stone passes out again, and he then finishes the act of micturition. However, in this case the patient distinctly tells us that the passage of water is of a fluttering character,—that is to say, he passes a certain quantity of water and then stops, and then goes on again, and this repeatedly. With a symptom of this kind one would expect to find that there is congestion of the trigone immediately around the neck of the bladder, and that there is a considerable amount of turgescence around the neck of the viscus. This is what you generally find when you have intermittency of flow in connection with tubercle of the bladder. Cystoscopic examination, however, has not revealed any such symptom, and I confess I am unable to account for the fact that he has got intermittency of this kind, unless it is of a reflex character. Incontinence of urine is a symptom which you sometimes find in connection with tuberculosis of the bladder. You find this particularly in women, and it occurs when the disease attacks the trigone and around the neck of the bladder. Of course an examination of the urine is made, and it is found to be turbid. The urine in connection with tuberculosis, whether of kidney or bladder, is of a peculiar character. Whenever you see it in the glass you can suspect you have to deal with tubercle, because it is of a turbid, flocculent character, and you will find that the sediment takes a long time to settle down; it does so very slowly. On examination it is found that there is pus in the water, and if the urine is alkaline you will find there are phosphatic crystals. But the important point is to ascertain if the tubercle bacillus is present. It is sometimes very difficult to find the tubercle bacillus. In cases of this kind it may be necessary to have recourse to cultures or to inoculations of guinea-pigs or rabbits.

With reference to the prognosis, in these cases I need scarcely tell you that it is always very grave. Tubercle of the bladder is extremely grave, so is tubercle of the kidney; but in the case of the kidney I should say it is not so bad as in tuberculosis of the bladder, particularly if the disease is confined to one kidney. In the case of involvement of one kidney only I have had several successful removals of that organ. But the bladder is scarcely a viscus which you can attack wholesale, and I am sorry to say that surgical interference with these cases is not often successful.

Digitized by Google

In tubercle of the bladder the important point is to treat the patient on general principles, to treat him as if he had tuberculosis of the lungs or of any other part. That general treatment should always be directed to increasing his strength, to try to enable him to throw off the disease. There are cases on record, and I have seen such myself, in which patients suffering from tubercle of the bladder have been able to cast it off, and I hope that under proper treatment this patient will be able to do so. Part of that treatment will consist of giving cod-liver oil, iron, strychnine, and guaiacol carbonate, or benzoate of guaiacol, because benzoate itself has an important action upon the bladder. In a case of this kind you have a great deal of pus present, and the drug which has the most beneficial influence in stopping the pus is urotropin; indeed, that may be regarded as our sheet anchor in tuberculosis of the bladder. A great deal was written some time ago about direct interference by injections in these cases of tubercle of the bladder, but unfortunately injections are of little avail in the vast majority of cases. Our sheet anchor in cases of general cystitis is nitrate of silver, but it has been found that nitrate of silver injections are absolutely useless in tuberculous ulcer of the bladder. Iodoform emulsion was also very much resorted to at one time, one and a half ounces of it being injected into the bladder, and left there until the next act of micturition. The results of this also, though applauded at the time, were, unfortunately, found on more close examination not to be very successful. But the drug which has been found most useful in these cases for injections into the bladder is the corrosive sublimate, commencing first with a strength of 1 in 5000 or 6000 in water, and then gradually increasing the strength to 1 in 2000. In passing the catheter in all cases of tuberculosis of the bladder you must be extremely careful not to introduce a septic instrument of any kind. That is a caution which applies to all cases of disease of the bladder. If that caution is not acted upon cystitis will probably follow, and the state of that patient will then be terrible indeed. Therefore having rendered your soft catheter aseptic, you pass it into the bladder and draw off the water, and then inject one and a half ounces of this corrosive sublimate solution and allow it to flow off at once. This process should be repeated four or five times, never throwing in more than one and a half ounces of the solution, because in all cases of ulceration of the bladder it is found that if you distend the viscus by a large quantity of fluid there is an increase of the symptoms.

The final procedure to have recourse to in such cases as this is cystotomy, either by the perinæal or the supra-pubic method. The perinæal method has now been practically abandoned, because by this route one is unable to see the state of the bladder; and in the second place, as tubercle is generally confined to the base of the bladder, whether at the trigone or round about the ureteral orifices, any instrument which you place in the bladder through the perinæum for drainage would come in contact with these ulcers and cause great irritation. Besides there is danger of the tubercle extending to the wound. But the principal reason for having recourse to the supra-pubic operation is that you can by this route not only feel the state of the bladder but you can also see it by means of a speculum and forehead lamp. There are certain cases, undoubtedly, in which, when the disease is localised good results have been obtained by scraping these ulcers by means of a sharp spoon, and then rubbing the part with iodoform. In very severe cases of this disease, where there is great strangury and a constant desire to pass water, and in which the bladder is very much involved, it may be necessary to open the bladder for drainage, particularly when the disease is complicated with cystitis. In such cases as this sometimes good results are obtained by draining the bladder supra-pubically. But in a general way I am afraid that in any extensive tubercular disease of the bladder we cannot look forward to anything more than what may be called temporising or palliative measures, whose immediate object is to ameliorate the sufferings of the patient.

Modified Treatment of Typhoid Fever.—Dr. T. B. Greenley advocates the use of acetanilid with quinine, which, he claims, has a soothing and quieting effect, and prevents the possibly irritating effects of the latter. He has never observed any depressing effects of the drug upon the heart. The dose is increased or diminished according to the rise or fall of the temperature.—*Med. Record*, June 15th.

Digitized by Google

PRESENT DAY SKIAGRAPHY.

WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL.

Demonstration III.

Gentlemen,—I will to-day pass on to the second part of my subject, viz. the diagnosis of aneurysm. Of all the various cases of chest disease to which the X-rays may be applied for the purpose of diagnosis—and I had almost said prognosis,—there are none in which they are found to be of greater use than in the diagnosis of thoracic aneurysm. So much is this the case, that I think we are justified in going as far as to assert that it is the duty of the physician to make use of this method of diagnosis in all doubtful cases of chest disease, especially if from the signs and symptoms an aneurysm is suspected. But here a word of caution is necessary. In examining a chest we frequently see a small oval bulging to the left of the aortic shadow, which easily to the uninitiated might be mistaken for an aneurysm. The shadow of this bulging of the aorta is frequently found in strong athletic men; it is far less commonly

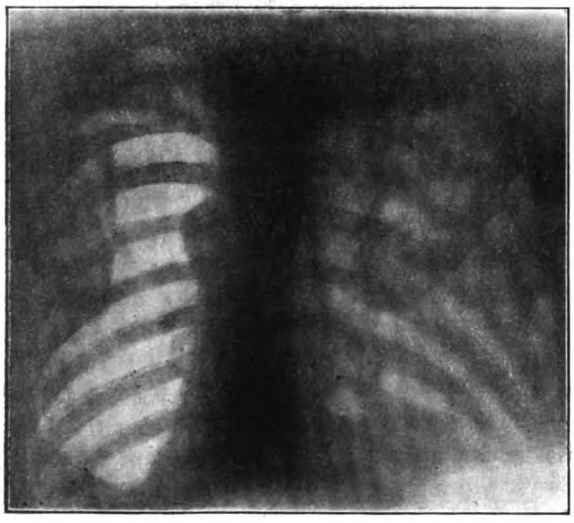


Fig. 1.—Oval bulge seen to the left of the aortic shadow. The chest is viewed from the back.

seen in women. It is never of very large size, and certainly may be disregarded, as I have seen it so frequently, especially in chests where I had no reason to suspect there was any aneurysmal dilatation. Before giving a case of thoracic aneurysm, I should like to say a few words upon the position of the heart in this disease. On more than one occasion my attention has first

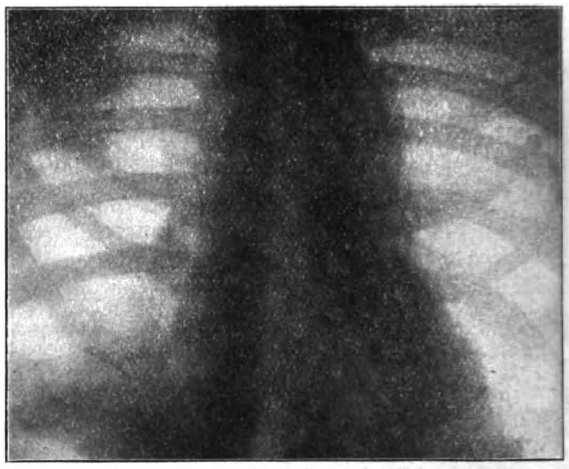


Fig. 2.—Showing aneurysmal sac and transverse position of heart. The chest is viewed from the front.

been called to the probable existence of a thoracic aneurysm by the transverse position taken up by the heart. It would appear as if the organ were pressed into this transverse position by the supra-incumbent weight of the aneurysmal sac (Fig. 2).

The two following illustrations are from the chests of patients in whom the aneurysm was first detected by the Röntgen rays. One was a gentlemen forty-two years of age, who was sent to me by my colleague, Dr. Heron, for an X-ray examination of the chest. The case was a doubtful one. The patient had been under the care of two or three physicians. He suffered with severe intercostal neuralgia, and some tenderness over the upper dorsal spines. There were no other symptoms or signs of aneurysm. With the screen examination, however, it was at once seen that this patient had a large aneurysm of the descending thoracic aorta. The following skiagram shows the chest of this patient (Fig. 3).

The next case was that of a woman fifty-one years of age, who I admitted to the hospital as long ago as 1897 with well-marked signs of aortic regurgitation. She remained in the hospital for eight weeks, and was discharged somewhat improved. She returned to hospital in December last, saying she had been in very much the same

Digitized by Google

condition with one exception, and that was the pain in the back between the shoulders had markedly increased. She described it as if the spine were being scratched with a broken piece of

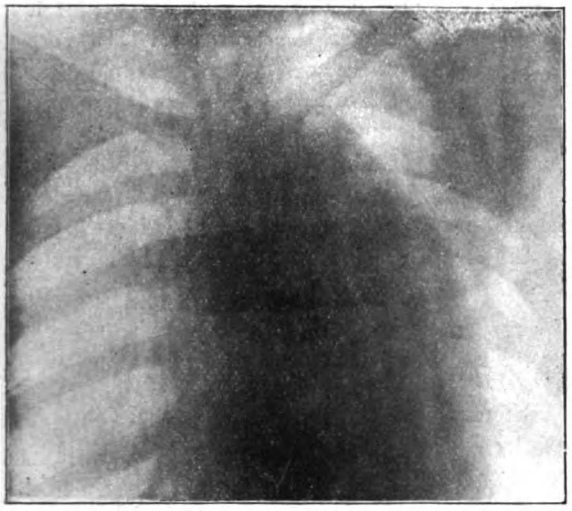


FIG. 3.—Large aneurysm of descending thoracic aorta. Chest is viewed from the front.

glass. As the X-ray apparatus had been installed in the hospital since the patient's first visit, I at once examined her with the screen, and had no difficulty in diagnosing an aneurysm of the descending thoracic aorta. The following radiograph

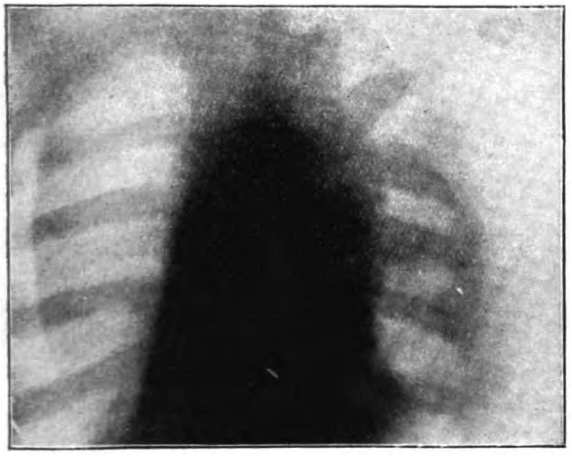


FIG. 4.--Aneurysm of descending thoracic aorta. Chest is viewed from the front.

shows this patient's chest (Fig. 4). Of course it is an open question whether this aneurysm existed as long ago as 1897 : all that I can say is that then, as now, there were no signs or symptoms of its presence with the exception of the pain in the back. I think there can be no question that when the Röntgen rays come into general use in clinical diagnosis, that aneurysms of the thoracic aorta will be diagnosed very much earlier than they are at present, and I think we shall find that the life of an aortic aneurysm is possibly very much longer than we now suppose.

The next case is an aneurysm (see Fig. 5) of the aorta, and I give it because it well illustrates that the rays may possibly be of use not only in diagnosis,

FIG. 5.—Large sacculated aneurysm of transverse arch of aorta, showing the marked difference of density between the shadow of aneurysmal sac and the heart.

but in prognosis. It will be seen from the skiagram that the shadow of the aneurysmal sac is very much darker than the shadow of the heart, which must mean that the aneurysmal sac contains a considerable amount of blood-clot, and therefore shows up in marked contrast to the fluid blood within the heart. This may ultimately become an element in prognosis, since from the density of the shadow we can approximately judge the amount of laminated clot within the sac of the aneurysm. This illustration was kindly sent to me by Mr. Payne.

The next radiograph is from the chest of a gentleman forty-seven years of age. I was asked

Digitized by Google

to skiagraph the chest for thoracic aneurysm. It shows very well the sac of the aneurysm, which was of large dimensions, and I merely give the case because it was followed up to its termination, and one had an opportunity of seeing the specimen after its removal from the thorax.

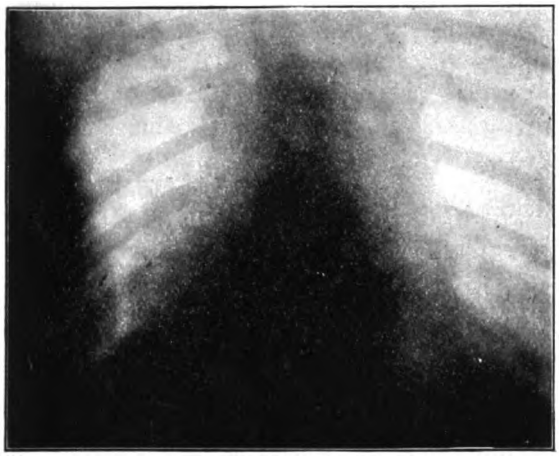


FIG. 6.—Aneurysm of transverse arch. Chest viewed from the front.

I will give another case of unsuspected aneurysm which was first discovered by the Röntgen rays. A woman, forty years of age, was sent up from the country to the Victoria Park Chest Hospital with the diagnosis of laryngeal phthisis. She was admitted under Dr. Beale. On examination with the laryngoscope, paralysis of the left vocal cord was discovered, which, of course, in itself was extremely suspicious of aortic aneurysm. There were no other signs or symptoms of aneurysm, however, so the chest was radiographed. The following is the picture of this patient's chest, showing clearly a large aneurysm at the junction of the transverse with the descending portion of the aorta (Fig. 7).

I will now give an example of probable cardiac aneurysm. I say probable, because the patient is still under observation, and cardiac aneurysms are confessedly of rare occurrence. I first saw this patient, aged thirty-three, over two years ago. He came to the hospital complaining of a cramp-like feeling in his chest, which extended down his left arm. There was a specific history of fifteen years ago. He had never had rheumatism or rheumatic fever. In the third intercostal space, three and a half inches from the middle line, there was noticed an obscure area of pulsation, over which there was a loud rasping systolic murmur. The same murmur was heard at the apex immediately following the first sound. Suspecting an aneurysm of the aorta, I took a skiagram of this patient's chest, and it was at once seen that the heart was of a very peculiar shape; there was a projecting shadow from the left base of the organ, corresponding almost exactly to the position of the left auricular appendix, and if aneurysms of this chamber of the heart were not extremely rare, there being, I believe, only a few on record, one would think from the skiagram that the patient had an aneurysm of his left auricle. The patient was admitted to the hospital under the care of Dr. Eustace Smith, where he remained for eight weeks. From the City of London Hospital for Diseases of the Chest he went to the Middlesex Hospital, where he was also admitted and remained for some time. About a year afterwards he returned to the Victoria Park Hospital, saying that he had come back because his breathing was getting shorter, and three days previously to his coming to the hospital he fainted in the street. On examination, the physical signs were, as far as I could make out, practically the same as they were at his first examination, with the exception that there were now subcrepitant

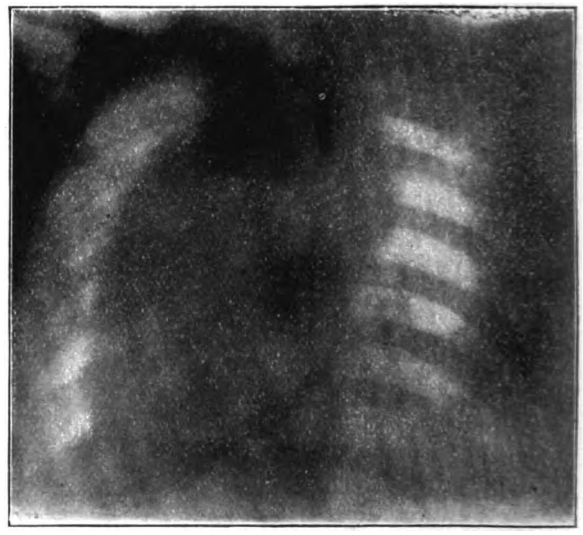


FIG. 7.—Aneurysm of descending and transverse aortic arch. Chest viewed from the back.

Digitized by Google

râles heard over the left posterior base of the lung. I took another skiagram of this patient's chest (see Fig. 8), which shows that the tumour at the left base of the organ has increased to nearly double its original size. With the screen there was marked pulsation in the tumour. I have taken a third,

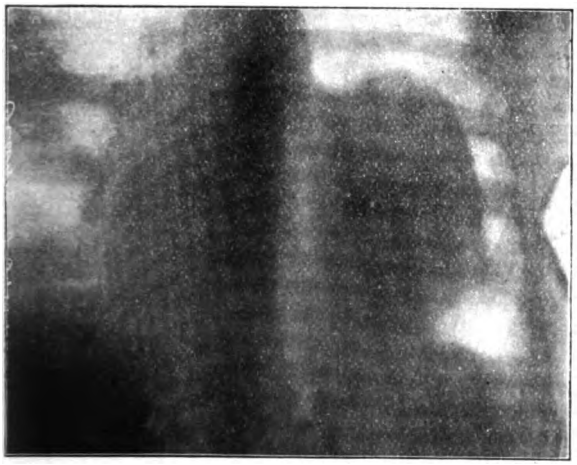


FIG. 8.—Probably an aneurysm of the left auricle. The chest is viewed from the front.

and even a fourth, skiagram of this patient's chest, and the projection from the left base of the heart goes on steadily increasing. He came to my out-patient room last week, saying that he was in much the same condition, with the exception that he had now begun to have a little hæmoptysis.

A Case of Barlow's Disease.—Dr. G. Variet reports a case of infantile scurvy with pseudo-paralysis of both legs, in a boy of 7 months (*Bulletins et Memoires de la Societe Medicale des Hopitaux de Paris*, 1901, No. 8). The child had been taking Gaetner's milk, which is cow's milk with half its casein deducted and lactose added, since the age of two months. He had no teeth. Upon diluted unboiled milk and orange juice, the conditions cleared in two weeks. After a full review of the literature of the subject, Variet concludes that Barlow's disease bears no relation to adult scurvy; that it is probably hæmorrhagic rickets; that it is very rare in France, though sterilized milk is very generally used; that modified milk rather than pure milk causes it; and that it is not a serious condition, as change of diet will effect a cure.—*Philadelphia Med. Jour.*, June 15th.

FOUR CLINICAL LECTURES
ON
SOME AFFECTIONS OF THE KIDNEY.

Delivered at Westminster Hospital.

By A. H. TUBBY, M.S., F.R.C.S.

LECTURE IV.

GENTLEMEN,—The subject to-day is hydronephrosis, and we should be clear as to what this means. It is dilatation of the pelvis of the kidney and of its substance owing to an obstruction to the flow of urine. Of course the amount of dilatation varies very much. It may be slight or it may be very great. Dilatation is also in some cases distinctly intermittent.

As to the causation: hydronephrosis may be either congenital or acquired. A very considerable number of the cases are congenital. Any cause which blocks the ureter or prevents free entrance of urine into or free exit from the bladder through the urethra is apt to give rise to a condition of hydronephrosis. Among the congenital causes there are twists or kinks in the bowel or in the ureter itself. There may be distinct stenosis or narrowing, or even actual obliteration of the ureter, or you may have pressure on it exerted by abnormal arteries. All those causes which tend to obstruct the flow of urine give rise to congenital hydronephrosis.

The acquired causes interest us rather more than the congenital; and of the acquired the most common is the presence of stone in the ureter or in the pelvis of the kidney. A small round stone which is able to move about freely, will sometimes become lodged in the upper part of the ureter, and will act as a ball valve, and it is just in those cases that you have an intermittent cystic swelling of the kidney, which one may describe by the term intermittent hydronephrosis.

Another condition which gives rise to hydronephrosis is movable kidney or floating kidney. The ureter becomes distinctly kinked and temporarily occluded, so that the urine will accumulate in the pelvis of the kidney, producing dilatation of the pelvis and kidney substance. Again, peritoneal bands or adhesions may cause it. In women, after movable and floating kidney, perhaps the

Digitized by Google

most common cause is pelvic tumour, especially cancer of the cervix uteri. The cancer grows around the cervix, and invades the posterior inferior aspect of the bladder, and gradually presses on the ureters so as to give rise to hydronephrosis. In the male there may be tumours of the bladder and prostate responsible for hydronephrosis. Enlarged prostate has the same effect, for it means obstruction to the flow of urine from the bladder, and there is dilatation of the bladder, and secondarily of the ureters, and finally of the pelvis of the kidney. Stricture of the urethra also gives rise to it. I used to question if it were possible for stricture in the urethra to cause dilatation of the kidney, but there is no doubt of it. Prolapsus uteri has been known to cause it, especially total procidentia, that is to say where the wall of the uterus is external to the surface of the body. And in these cases the bladder is dragged down with the uterus, and the ureters become kinked, so that there is retardation of the flow of urine. Injuries to the ureter will also cause hydronephrosis, and so, in the male, will cancer of the rectum. Thus there is a good list of causes for you to remember. Some of them are chiefly of pathological interest, but many of them are of extreme clinical interest. Those possessing the greatest importance are calculus, movable and floating kidney, enlarged prostate, stricture of the urethra, and, in the female, cancer of the uterus.

Hydronephrosis, if it is due to a cause affecting the bladder, involves both kidneys. If, on the other hand, it arises from a unilateral cause, it affects only one kidney. So hydronephrosis may be either bilateral or unilateral. In 33 per cent. of the cases it is bilateral.

With regard to the changes which are produced in the kidney by backward pressure, one may sum them up by saying that the kidney substance undergoes partial atrophy and fibroid change, with a final result that the secreting substance of the kidney is slowly and definitely destroyed. There is destruction of tubules with excessive fibroid change in the tissue; so that in what is left of the substance of a hydronephrotic kidney you have changes which are very closely comparable to that seen in contracted granular kidney. It is a truly fibroid change.

The first thing which happens where there is backward pressure is that the pelvis of the kidney and the calices become dilated. I take it that in the healthy kidney there is no actual space in the pelvis or calices, but that what room exists is made by the passage of a few drops of urine between the opposed surfaces. In hydronephrosis, when fluid is retained, the first thing that happens is that the pelvis of the kidney is dilated, and that is followed by dilatation of the calices. There are a considerable number of calices branching from the pelvis. If you look at a normal kidney you will find that the pyramids push themselves forward in a wedge-shaped manner, and come to an apical point in the calices, so that the normal calices are filled up by pyramids. When hydronephrosis sets in, these pyramids become smaller and more obtuse at the apices; then they become flat, and finally, from being apical in one direction, they become concave in the opposite direction. That will give you some idea of the amount of pressure present when it will produce such a result as that. At the same time the kidney increases actually in size, but not in weight, so that the intervals between the pyramids become greater and greater; the pyramids become concave-surfaced structures, separated by a considerable space of renal tissue and by the remains of the columns of Bertini. Externally there is the renal cortex or true secreting substance distended and much thinned, so that the cortex, the most important part of the kidney, may be only one line in thickness, instead of seven or eight lines as normally.

You will readily understand that with all this pressure and stretching there must occur in the kidney some very profound change, and that profound change is the one I have already indicated to you. One case is described by Dr. Hilton Fagge, in which the renal tubules, instead of being straight when looking towards the pyramids, were distinctly zig-zag, showing the tension which they had undergone. The calices all this time have been undergoing changes; they have not only shared in the general dilatation of the pelvis of the kidney, but have undergone the definite dilatation, and are oval and egg-shaped, and communicate with the pelvis by a narrow rounded aperture. The tumour enlarges, and may attain such a size as to be mistaken for ovarian cyst. I have seen this error made, for the hydronephrotic kidney may occupy a large portion of the abdomen. You may avoid mistaking an ovarian cyst for hydronephrosis by noting that the

Digitized by Google

latter tumour is kidney-shaped. What is the nature of the fluid in the cyst? It appears to be urine, but it is anything but true kidney-secretion. Often it contains no urea nor uric acid; the constituents of urine, as a matter of fact, have all but disappeared. The secretion is a clear watery fluid of low specific gravity, and contains a trace of albumin, and it is difficult to explain why it should do so. It contains sodium chloride also. Occasionally, however, the fluid is brown in colour, which is due to the admixture of blood with it. Or it may be thickened and colloid, or even be ammoniacal. If pus is present there is produced a new condition which is known as pyonephrosis.

Now a few words with reference to the *symptoms and signs*. The most striking thing is a tumour which is renal in character, and appears first of all in the loin. It has no pelvic connections, and it does not rise up from the pelvis. On examination *per vaginam* or *per urethram* you cannot find any connections to it. Unless it is very large it does not move up and down on respiration, and colic resonance cannot be elicited behind nor at the side of it, but is well in front of it. There is no resonant intestine in the loin. It has all the characteristics of renal swelling—fluctuating, rounded, and, as a rule, uniform though with exceptions. In some cases dilatation of calices is so much greater than the dilatation of the pelvis that you find the kidney lobulated in outline, and it is those cases which are mistaken for cystic kidney.

Then the tumour fluctuates in size. You must watch such a tumour and note any alteration in the quantity of urine passed. If the patient is observant, he will tell you that he suddenly passed a great quantity of clear urine before the swelling subsided somewhat. When the patient does pass that clear watery urine there is much less urea in it than normally, but there may be a little albumin.

As to pain, this is not a very reliable symptom, because it is often absent. If it is severe and paroxysmal it generally means that the hydronephrosis is due either to stone or else to pus. Lastly, there is little constitutional disturbance unless pus be present.

As I have said, the diagnosis is often difficult, because of the frequency with which the tumour may be mistaken for ovarian cyst in women. But that is a matter of careful examination. You ought to explore digitally the pelvis and the rectum and vagina, and obtain a clear idea as to the origin of the swelling. Hydatid cyst will also give rise to difficulty in diagnosis, and it has often done so. The last refuge of the destitute in diagnosis in abdominal swellings is hydatid cyst. Sometimes this "comes off" as a brilliant diagnosis. But you should endeavour to confirm it by seeking for daughter-cysts in the urine. Hydronephrosis has also been mistaken for ascites. I mention ascites, because such an error, if made, is a very serious one indeed. Hydronephrosis may be also mistaken for renal cystic disease. This mistake may be justifiable if there is lobulated hydronephrotic kidney. You had better not try to remember all the points of difference, but carefully note the cardinal points of hydronephrosis.

As to the *prognosis*, this depends upon whether the disease is bilateral or unilateral. If the disease is due to some cause in the bladder which is non-removable, or of a malignant nature, and therefore of an intensely obstructive nature, the prognosis, of course, is essentially bad. Or if the disease has been allowed to go on for a long time, so that a great deal of kidney substance has been destroyed, then the prognosis is also bad. If, on the other hand, the origin of the hydronephrosis is unilateral, and dependent upon a cause which is amenable to treatment, such as a movable kidney or stone, then the prognosis is by no means hopeless. It often happens that the healthy kidney takes on a hypertrophic growth, and performs the work of both kidneys; in fact, it is a very common thing for one kidney, when the other is thrown out of gear, to grow and attain double the normal size, and do all the work required. There is one other drawback; sometimes the mistake has been made of removing the healthy kidney, and leaving the diseased one behind. There is a very fruitful field of investigation open to anyone who can discover a means of finding how much urea comes from one kidney, and how much from the other.

Treatment.—In the case of unilateral hydronephrosis or bilateral hydronephrosis, unless the tumour is giving rise to discomfort and pain it is better to leave it alone and watch it. If it is very large you may interfere, but if the patient is experiencing no pain, and the cause is a non-removable one, it is better to leave the kidney alone altogether, even though it may be large. Some physicians have advised a method of com-

Digitized by Google

bined friction and compression of the tumour, by virtue of which something may be disturbed and dislocated, and the urine suddenly passed down the ureter. It seems to me that that is very dangerous advice. That method may cause urine to be discharged into the peritoneal cavity, with fatal results. It is a mode of dealing with an affection in the dark. If the swelling should continue to increase in size it is advisable to tap the kidney. It often happens that one tapping is not sufficient, and then the tapping should be repeated three or four times. Sometimes the tapping is effectual, and the patient goes on well. In the absence of a post-mortem examination, it is difficult to say how far tapping has been successful, and what has been dislodged from the ureter so as to allow the urine to flow in a normal way.

We must now ask the question where we should tap, and how? If you do it on the left side, that is to say, on the side in which the liver is not so largely placed, you may tap one and a half inches below the tip of the last rib, and outside it.

If it is on the right side you are in great danger of wounding the liver if you tap at the corresponding spot. You should puncture at the midpoint of a line which is drawn from the tip of the last rib to the anterior superior spine of the ilium, and about two and a half inches behind the anterior superior spine. It is most important that when you tap you should observe the utmost aseptic precautions, because it has happened that hydronephrosis has been converted into pyonephrosis simply from the want of them. The skin should be thoroughly sterilised, and the instruments boiled, and rigorous asepsis observed. The operation does not simply consist in plunging a trocar into the kidney, and letting the fluid run out of it. You should have a proper aspirator, which should be sterilised, so that no air is admitted into the kidney. Greig Smith, in his work, advised that the trocar itself should be filled with fluid, so that you do not introduce any air into the kidney, and therefore you will probably introduce no septic organisms. He gives it as his experience that cases of hydronephrosis have been converted into pyonephrosis by lack of antiseptic precautions.

The next question is, supposing you tap repeatedly and the tumour begins to grow again, should you perform nephrotomy and drainage? Yes. Remember that nephrotomy is cutting into the kidney substance, nephrectomy is removal of the kidney, nephrolithotomy is removal of stone, and nephrorrhaphy is sewing the kidney to the posterior abdominal wall. Nephrotomy has been practised, but unfortunately it is rarely curative unless at the same time you can remove the stone. If this measure fails, you must consider the question of nephrectomy. This operation, which is a severe one, is done when nephrotomy and tapping have failed. It is also performed if the amount of urea passed day by day is considerable—that is to say, from 350 to 400 grains per ounce, but not less. Finally, nephrectomy is done if the kidney is found so hopelessly disorganised that it must be a source of danger if left.

We now come to pyonephrosis, which I have already defined. Pyonephrosis is usually associated with either stone, gonorrhœa, stricture, or enlarged prostate. That is to say, there is a kidney which has been the site of hydronephrosis, and has been infected by septic organisms. There are plenty of septic organisms with gonorrhœa ready to travel backwards. How does the kidney become affected? There is no doubt it is through the lymph-channels—that is to say, pus or septic organisms make their way up to the kidney by these channels and infect it.

The *symptoms of pyonephrosis* are those of hydronephrosis plus those of abscess. But the pain is much greater, and there are œdema and tenderness in the loin. Pus is occasionally present in the urine, especially when the urine has been discharged suddenly in large quantities. With pyonephrosis there are constitutional symptoms of septic infection.

The *treatment* is either nephrotomy or nephrectomy. Pyonephrosis is met with mostly in association with bladder and urethral causes. It therefore follows that nephrectomy is not always a justifiable operation in many cases, because the other kidney is pretty sure to be in a state of hydronephrosis or pyonephrosis, and one has sometimes to content one's self with nephrotomy.

Formaldehyde.—The usual strength (ten to fifteen drops of a 40 per cent. solution to a pint of water) employed in a surgical wash should not be used about the eyes. The conjunctiva is too sensitive for a solution of such potency.—*The Canadian Practitioner and Review*, June, 1901.

Digitized by Google

REMARKS ON THE SUMMER DIARRHŒA OF INFANTS.

By J. D. WINDLE, M.D.

Medical Officer of Health, Southall-Norwood Urban District Council.

THERE can be little doubt that micro-organisms play an essential part in the causation of the summer diarrhœa of infants, by the brewing of intestinal toxines; and experience leads to the inference that they are introduced with the food, for in by far the large majority of cases the disease occurs in artificially-fed infants associated with a foul bottle, food unsuitable in kind, improperly prepared or administered—in the widest sense of these terms. In breast-fed children diarrhœa is often associated with a foul nipple shield (which is practically a "bottle") or a dirty "dummy."

There is one factor, viz. thirst, which has an important bearing on the subject of over-feeding, and thus becomes an indirect cause, the prevention of which is a valuable means of prophylaxis. Bottle-fed children are especially thirsty in hot weather for this reason; their food is usually given as concentrated as in cold weather. The thirsty infant takes the bottle ravenously; in its mother's phraseology, "the food does not satisfy it," so it gets over-fed from a greater concentration or amount, and an acute attack of diarrhœa and vomiting ensues. Over-feeding from thirst (apart from contamination of the milk as mentioned above) is probably the sole cause of diarrhœa in *breast-fed children*, and is a comparatively rare event, for there is a natural provision against it, since the mother instinctively consumes more water in hot weather, and in consequence the secretion of milk is thinner and more plentiful; moreover, the first milk secreted during the act of suckling is more dilute.

Symptoms.—Broadly speaking, the severe types of the disease divide themselves into two groups; one in which the symptoms of toxic poisoning predominate, which is characterised by sudden onset—prostration, collapse—out of all proportion to the amount of diarrhœa and vomiting which may be but slight or entirely absent at first. This variety is well named *infantile cholera*, a definition which conveys a good idea of the phenomena observed. Such cases are often speedily fatal from exhaustion, coma, or convulsions; or reaction may ensue when the symptoms become much like those of the second type, in which the toxi element in the symptoms is still evident, but the most urgent are, diarrhœa and vomiting, and the general effects which proceed from these—intense restlessness, moaning, tossing of the head from side to side, and incessant movement of the lips and tongue, symptoms due to thirst. When there is abdominal pain, we find, in addition, acute cry, drawing up of legs, hardening of the abdominal muscles, sweating, and that pathetic, anxious, frightened look in the intervals of the paroxysms which is so painful to witness. Vomiting is constant, or all that is taken "runs straight through it," the motions varying in colour and consistence from chopped boiled spinach to dirty brown water. Respiration is quick, feeble, and shallow. The more acute the case the less the fever; coldness, pallor, and cyanosis, together with dilated inactive pupils, a coated cornea and stupor are harbingers of speedy dissolution, from coma and convulsions (a picture which has some resemblance to meningitis), or from exhaustion—heart failure.

Explanation.—The amount of water lost in the stools and by vomiting and perspiration is out of all proportion to that taken in; in most cases none is absorbed, vomiting being constant. The excess must, then, come from the blood and tissues. *Thirst* is the cause of the restlessness and moaning—symptoms often wrongly interpreted as due to physical pain. The enormous loss of weight, sunken fontanelles, flaccid skin, scanty micturition, dyspnœa, and cyanosis, are due to the same thing—dryness of the blood and tissues. The thick tarry blood stagnates in the right heart and venous system, the systemic pressure is lowered till it sinks to *nil*, and the heart ceases to beat.

Stupor, coma, and convulsions are due to the action of toxines on the nervous system.

Treatment.—The vital indication is to supply *water*, which at once relieves the most urgent symptoms, *thirst*—and vomiting—the blood-vessels of the stomach suck it up like a sponge; it refills the emptied vascular system, the blood pressure is raised, the heart regains its vigour.

The child will probably fight shy of the first teaspoonful—fearing probably it is beef juice or

Digitized by Google

medicine; but the next, and all we give it up to half a pint—the greed—the hunger with which it is taken—the look of satisfaction—the calm—the sleep which ensues—tell us how badly it was needed.

Food and "stimulants" are harmful, as if they are retained there are attempts at digestion and absorption. Water supplies all that is essential, and the mucous membrane rests, recuperates, so that after twelve to twenty-four hours it is fit to resume light work. Moreover, in addition to relieving urgent symptoms, the water is directly curative—it dissolves the absorbed toxines, the kidneys again become active, and the toxines are eliminated by them.

The intestinal bacteria die from inanition—their pabulum is cut off—and should any escape they are not supplied with suitable material from which to brew their toxines. When the urgent symptoms have ceased—as they almost invariably will in twelve to twenty-four hours—what food are we to give? The natural food—milk, but in very dilute form; water must yet constitute the chief aliment. For the first day one part of milk to twenty of water, increasing the amount of milk from day to day until the normal proportion for the age of the child is reached.

The same principle applies to the treatment of the less severe forms of the disease.

Summary.—Stop food and stimulants of all kinds; give boiled cold water, as much and as often as the child will take. This exclusive water diet may be continued with perfect safety up to forty-eight hours, but it is only very rarely indeed that it will be required longer than twelve to twenty-four hours when diarrhœa and vomiting have ceased.

Tepid sponging if there is fever, hot dry packs if there is collapse, large linseed meal poultices to cover the abdomen if there is pain, are valuable auxiliaries.

HEROIN: A BRONCHIAL SEDATIVE.

By E. HAWKINS, M.A.(Oxon.), M.D.

In these days there are so many new remedies introduced, and usually with such high-sounding names, that the medical practitioner may be forgiven if he expresses his disbelief in them. Such a feeling, however, may lead to the neglect of many which are really valuable. The manufacturing chemist, realising that the crude drug is unreliable,—that the active principle, as first prepared, is possessed of qualities which in turn make it undesirable for administration—sets before himself the problem of preparing a salt of that active principle which shall contain its virtues in a constant and absolutely unobjectionable form. As there are always several experimenters striving for the same goal, it usually happens that several rival products will be upon the market at the same time, and experience alone will show which will survive. It is one of these salts, "Heroin," which I venture to think will live. Every medical man recognises the value of morphine in the treatment of the irritating cough of phthisis, it is found in many a linctus, and is one of the chief constituents of the well-known "Brompton-Mixture." But morphine has undesirable qualities, the chief of which probably is its constipating action, and its depressing action on the heart and on respiration.

"Heroin" is described as the di-acetic ester of morphine, and, as such, requires to be carefully distinguished from the acetate of morphia. The latter might be described as composed of four parts: (1) The hydrocarbon group $C_{17}H_{17}$, (2) the hydroxyl group of two hydroxyls, $(OH)_2$, (3) the nitro group NO, and (4) the acetic acid group, $C_2H_4O_2$ or CH_3COOH. In "Heroin" itself the fourth group is absent, as it is in morphia, and two acetyl groups replace the two hydroxyls, giving therefore $C_{17}H_{17}(CH_3CO)_2$ NO. This, not having proved sufficiently soluble for all purposes, has been further improved by the formation of a neutral soluble salt, the hydrochloride, $C_{17}H_{17}(CH_3CO)_2$ NO. HCl. Chemically speaking, the effect of the substitution is to convert an alcoholic group into an ethereal group, and thereby to obtain more rapid diffusion, but practically the change has produced a drug differing in many respects from the original morphine. "Heroin," although a sedative, does not materially reduce pain, nor does it produce sleep, except by allaying a cough which has caused sleeplessness.

The action of "Heroin" upon the respiration has been aptly compared with that of digitalis upon the heart, the whole is lengthened and strengthened so that the embarrassed breathing of asthma and phthisis are materially alleviated. The blood pressure is not affected, but the temperature is lowered to a greater extent even than after the administration of morphine. "Heroin" is much less toxic than codeine, whilst equally rapid in its effect. The dose is from one-twelfth to one-sixth of a grain, given in pill, or, as a powder with sugar, and the beneficial effects may be expected within half an hour. The hydrochloride, being soluble, is suitable for hypodermic injections, and the dose is the same, although it is advisable to commence with half the dose. I have used it with great success in phthisis, asthma, and bronchitis and have found it a valuable substitute for morphine in these cases.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 455. WEDNESDAY, JULY 17, 1901. Vol. XVIII. No. 13.

CONTENTS.

PAGE

*A Clinical Lecture on the Chorea of Pregnancy. By AMAND ROUTH, M.D., F.R.C.P. 193

*A Clinical Lecture on the Treatment of Foul Wounds. By W. ARBUTHNOT LANE, M.S. 199

*Present Day Skiagraphy. WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL. Demonstration IV 203

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE ON THE CHOREA OF PREGNANCY.

Delivered at Charing Cross Hospital, May 25th, 1901.

By AMAND ROUTH, M.D., F.R.C.P.

GENTLEMEN,—I had intended to give a clinical lecture to-day on something quite different, but this patient came in a few days ago, and has given us the opportunity of talking about a somewhat unusual but very interesting combination of conditions, pregnancy and chorea. The only pity is that we have not had her under observation for a longer time. She has only been here a few days; otherwise we might have been able to notice further developments, which would have added more interest to the case.

Up to the 6th May, apparently, this patient had had nothing of a neurotic nature, such as we now find her the subject of, and she certainly had not had chorea. She is now five months pregnant, and only within the last fortnight has she had any symptom of chorea. It is evidently, at least I hope so, one of those mild cases of chorea, one of the transient varieties, which are the exception rather than the rule when occurring in pregnancy. She has already improved very much, but still one can see a good deal of irregular muscular movement.

I will just say a few words about the ordinary chorea, which is met with apart from pregnancy. Those cases which you have seen are mostly among the out-patients, probably not very severe, but every now and again there are severe cases which may lead to the patient being kept absolutely in bed, and which sometimes lead to a serious issue. The disease is almost entirely in children in the ordinary way, and in something like 75 per cent. of the cases the subjects are females. It may first occur at any age, but the attack which you see in the out-patient department is much more likely to be a second attack, for chorea may recur at almost any age after the first attack. A first attack of the disease is very rare after puberty. As regards its causation, we know that the children who thus

Digitized by Google

suffer are generally children of nervous families, that is to say, some of the members of a family may have had chorea, or epilepsy, or asthma, or some of the other more definite nerve storms. Somewhere about 30 to 40 per cent. of children with chorea have had, or have at the time, some rheumatic affection, generally articular, and very often one is able to find in the subcutaneous tissue some of those curious nodules which are called rheumatic nodules, in which the poison seems to be especially present. Another thing which is very often found in children with chorea is some variety of endocarditis. It is an exceptional thing not to find some murmur in the endocardium, generally in the mitral valve region, and it is so exceptional not to find it that the majority of people think that the endocarditis is in some way or another a part of the disease chorea. We shall have a little to say later on as to the probability of the association of the two. In addition to these predisposing causes of rheumatic troubles and endocarditis, associated with a nervous diathesis, there is no doubt that chorea can be induced by a strong nervous impression, such as fright or emotion, and it is probably also induced in a nervous child from observing the phenomena of chorea in a brother or sister, or in a schoolmate.

The main symptoms—and this applies equally to chorea accompanying pregnancy—may be grouped into four. First of all there are involuntary muscular movements of an irregular character. Secondly, there is inco-ordination during voluntary movement. Thirdly, there is weakness in the affected muscles. It is a question whether that weakness is really due to mischief—functional or otherwise—in the cortex of the brain, or whether it is due to the muscles being fatigued. I think very likely it is secondary to fatigue in the muscles from their constant movement. Fourthly, there is a curious mental disturbance in many of these cases, and that is more especially observed in cases connected with pregnancy. A cloudiness and dulness of intellect, sometimes amounting almost to stupor or to melancholia, can be observed. The involuntary movements themselves, or twitchings, as a rule begin in the face or arms, and generally the right arm, and then they spread, to a less extent, to the legs, and sometimes to the trunk. The mild cases come and go in a few weeks, sometimes a couple of months or so, and the severe cases will reach their acme in a week or two, and then retain that intensity for a few weeks, and then gradually tail off again. There are cases which persist, and cases which recur and never get quite well. The urine is not altered very much. It has been observed by numerous investigators that urea is very much increased; there is an excessive elimination of it. That, presumably, is from the waste of tissue from the muscle movement.

As regards complications, in the ordinary chorea the main complication is endocarditis, and in children who have had more than one attack of chorea there are very very few in whom you cannot detect some form of endocardial murmur. Those who believe that the chorea is due to a rheumatic poison, that it is a toxæmia of rheumatic origin, consider that the endocarditis is the result of that, and it seems likely that both the chorea, as we shall see later, and the endocarditis are the result of a toxic process.

Now a word or two about the pathology, before we consider the condition, with special regard to the co-existence of pregnancy. When I was a student very little was known about germs as a cause of disease, we were just beginning to think that germs were the cause of some diseases, but we were content with the delightful old theory that chorea was due to the mechanical effect on the cerebrum of minute flakes of lymph carried from the valves of the heart, and so causing irritation in distant parts of the body. The embolic theory of chorea was believed in thoroughly then, or perhaps I should say it was just beginning to be doubted. Bastian, and Hughlings Jackson, and others were beginning to think that emboli did not explain everything, and that there was probably some other causation. They tried to explain the condition of the brain by showing that it was due to thrombi that were formed in the smaller capillaries, and not to emboli. After a time different observers found different forms of germs present in the rheumatic nodules, in the joints, and in endocardial vegetations, and these germs were generally of the nature of cocci. More recently there is a paper, which I am indebted to Mr. H. S. Gabb, my house physician, for showing me, which is well worthy of your consideration, written by Dr. Poynton and Dr. Paine,* in which they describe how

* 'Lancet,' May 4th, 1901, p. 1260.

Digitized by Google

they have isolated and cultivated a diplococcus which they found in the joints and nodules, and in vegetations in cases of chorea. They have gone further than that, and with the help of other observers they have been able to show that these diplococci have been found also in the motor cerebral cortex, and that the changes which go on in the cortex are exactly those changes which take place in the joints and nodules, and in the valves of the heart, of course with the difference that nerve cells and other structures are involved, instead of merely connective tissue. They are able to show from what they have observed that the changes in the motor cortex are practically identical with the changes that go on in these other better understood structures. Further than this, by isolating and getting pure cultures of these diplococci they have inoculated rabbits with such a solution, and have been able to produce choreic movements in the face and limbs of these animals. So it is extremely likely we shall know sooner or later that chorea is really the result of the system being loaded with these diplococci. In addition to that, however, there is not the least doubt that all these diseases, due, primarily or otherwise, to cocci, are influenced to a great extent by the products of these cocci. The poisons which these diplococci give off cannot at present be estimated, but there is no doubt that a great many of the diseases which we cannot produce directly by inoculation could be produced if we could at the same time inoculate the secondary toxins produced by the disease. And I think if we consider chorea as it occurs in pregnancy, very often without the definite foregoing of a rheumatic disease, we shall see that the theory of a toxæmia is much more likely to be the correct one than that everyone of them is due to some specific germ. My own impression is that we shall find that the chorea of pregnancy is due, in some cases, to a rheumatic origin, perhaps to these diplococci which Drs. Poynton and Paine have described. But in other cases—and I think in the majority—it is due to some toxin which is of a similar nature as regards its effect, but yet is not identical with the rheumatic poison. That, however, we cannot be very definite about yet. But I think it is pretty clear now, for instance, that diphtheritic paralysis is not due to the germs which produce diphtheria, but to some poison which those germs have produced in the system. That is to say, it is a toxin secondary to the infection, and the probability is that we shall find something of the same sort later on as a cause of chorea. The great argument against chorea being the result of a toxæmia is that it is sometimes unilateral; that is to say, it may be a hemi-chorea, and that, as in this girl, is not an uncommon thing. But it almost always migrates to the other side, and may become generalised. It is not unknown for a patient who is known to be toxæmic to have local manifestations, because one simply has to assume that the diplococci have, for some reason or other, infected a particular area of the brain on one side instead of both sides. The affection ought to be bilateral, but there are plenty of instances which one could enumerate in which the poison is temporarily localised.

Now to come to the chorea of pregnancy. There are two or three things I have not alluded to in the general chorea which we must mention in connection with the chorea of pregnancy. First of all, the symptoms are practically the same in the chorea of pregnancy and in the ordinary chorea. The physical evidences are similar to those we have already described. The onset is generally within the first two or three months, certainly in the first five months of gestation. And it is generally in *primæ gravidæ*, and proportionally more frequently in the unmarried. The women who are the subjects of it are young. One expects that, because the women are usually, as I have said, *primæ gravidæ*, and cases are seldom seen over twenty-seven years of age. Now the rheumatic poison or the presence of any articular rheumatism is found in only 30 per cent., and about 33 per cent. have previously had chorea. The great majority of those 33 per cent. have rheumatic evidences, so that these two classes largely overlap, and we have practically something like 60 to 70 per cent. of cases where there has been no rheumatism and no evidence of previous chorea. It is difficult to explain chorea under those circumstances, and I think that that is one of the strong arguments in favour of the chorea of pregnancy being due to some toxic condition other than rheumatism. One of the strongest arguments in favour of that theory is that, however bad the case of chorea in pregnancy, if it be taken early enough, if you end the pregnancy—that is to say, bring on abortion, the

Digitized by Google

chorea ceases. The onset of many of these cases of chorea in pregnancy seems to be some nervous shock. Sometimes the discovery of the patient that she is pregnant is sufficient to bring it on. At other times there is some other form of shock. It must be remembered that during pregnancy the nervous system of the patient is, as it is called, very unstable. Pregnant women suffering from uræmia or toxæmia with albuminuria are more liable to get eclampsia than other people who are not pregnant. Several cases of chorea are reported to have come on when the patient first feels the child moving,—in other words, when she quickens. This girl says she quickened a month ago; I do not know whether that is quite correct, because she is only five months pregnant now, and it is not usual to quicken or feel the sensation of the child's movements until the eighteenth or twentieth week. But she seems to be fairly clear on the point herself. If she had quickened at her usual time, that is to say, a fortnight ago, it would have occurred at much the same time (May 6th) that she began to have these twitchings. This patient has no need to be distressed at her pregnancy. Therefore, probably, the fact that she was pregnant would not operate as a determining factor in her case.

We know a few things about chorea in pregnancy. We know that if a woman gets over one attack she is apt to get another attack at her next pregnancy, and that each attack seems to be a milder form. It is generally a bilateral chorea, and the mental confusion associated with it is much more marked than in ordinary chorea, probably because most of those cases are those where the mental condition is unstable, and therefore the different varieties of mental disturbance are more apt to occur. Another thing noticed, and which was very marked in the second case which I shall draw your attention to, is that as the chorea passes off there is often some mental phenomenon supervening. That has been observed almost constantly in severe cases. It is apparently a transference or variation of the particular variety of nerve storm which is going on. Now, if the type is severe, it causes a good deal of disturbance to the patient, for two reasons: first of all the subjects of it cannot sleep; their twitchings keep them awake, and naturally their general health, and especially their nervous system soon suffers. Secondly, they cannot take food; they cannot feed themselves, and they cannot keep still to have the food given to them, and they therefore get more and more exhausted. Thus, in really bad cases the mortality is as high as 20 to 30 per cent. —I am talking of cases during gestation—and the explanation is not that it is due solely to this exhaustion of which I have spoken, but that it is in great part due to the tendency there is for septic changes to take place during the premature labour. If the exhaustion is great and the muscle movements well marked, as the result possibly of the blood being charged with urea, or as a result of the intensity of the toxin which is producing the whole disease, the fœtus very often succumbs, and many cases are recorded in which premature labour has been induced and the fœtus has been found *in utero* in a state of decomposition. The result of such a condition is that the patient suffers from septic intoxication, which in one sense, is an auto-toxæmia; and that seems to explain not only the tendency to septic changes, sapræmia or septicæmia coming on during the premature labour, but also to the mental phenomena which so many observers attribute to auto-intoxication.

I will now read you a few notes of the case which my clinical clerks, Mr. Hood and Mr. Capp, have drawn up for me about this particular patient. Though it is not a very typical case, you will see the significance of some of the symptoms. The father died from some cause unknown. The patient had one half-brother on her mother's side who had St. Vitus's Dance when a child. So there is a distinct family history of the same trouble. This girl is one of twelve children, six of whom have survived. We know nothing of the cause of death of the other six. This patient has had nothing very much wrong with her as a child, except measles. She is known to have been of a nervous and rather fidgety type of character, but had never had any attack of rheumatism or chorea at all. She began to menstruate at fourteen and a half years of age, and everything has been quite regular since, but for two or three years she has been anæmic and rather out of health. She was married on the 15th December last, but was unwell almost immediately afterwards, ceasing to be unwell on the 24th December. Since that date she has not menstruated. That shows the gestation to be at about the twenty-first week. She

Digitized by Google

dates this particular attack of chorea from about 6th May, when she had a slight twitching of the right arm and right leg. This gradually got worse until she saw Dr. Mott in the out-patient department, and she was by him encouraged to come in. She declined at first to do so, but subsequently she did come in on the 16th of this month, and was then in a fairly bad condition. Still the disease was entirely unilateral. The right arm was twitching constantly, and the right leg to some extent; but under the influence of bromide (ten grains) and rest in bed, with a screen drawn round her so that she has not been worried about anything, she has very definitely improved. Voluntary movement seems to have quite recovered itself. You can see that she has very fair control over the muscles now, but previously, as she lay in bed you could notice the involuntary movements of the trunk muscles even, especially on the right side. Even now her right arm is not held up above her head as steadily as the other, and twitches considerably when lying passive. The reflexes are not altered in these cases, or at all events only slightly. Gestation is proceeding normally; the uterus is just below the level of the umbilicus.

The only thing noteworthy about her past history is that she has teeth rather suggestive of congenital syphilis, and she has also some curious linear scars round about the mouth, as if there had been sores there. I do not know whether that is of any practical significance, for syphilis is not supposed to have any direct association with chorea. As you see, it is a very mild case, and she is already getting better of it, though there is a little trace of choreic movement in the left hand today, which may mean an impending migration of the movement. Dr. J. W. H. Eyre has not succeeded in finding any germ in the blood of this patient.

Here are the notes of another case which also occurred in this Hospital, under the late Dr. Pollock. The case was that of a girl æt. 20, unmarried. Three days before admission she developed suddenly choreic movements, and became delirious at night. On admission there were violent bilateral choreic movements, a dull intellect, indistinct articulation, but no pyrexia, no albuminuria, no cardiac disease, and no paralysis. There was no previous history of chorea, of rheumatism, or of fright. The movements were worst above the waist. Taking food and sleep were alike impossible. She speedily became exhausted, and lay in the typhoid state, but there was no cessation of the movements except under chloroform, all other drugs proving useless.

That leads me to speak of treatment. In ordinary chorea there are plenty of drugs which do soothe the patient, and they enable her to tide over the trouble; but when it is a bad case of chorea in pregnancy, absolutely no drug has the least effect as regards cure. In the mild attacks you can get the patient to sleep by means of giving chloral or conium, and it can sometimes be procured by heavy doses of bromide. But that is merely a temporary measure, and the patients wake up out of the sleep as bad as ever. In the very severe cases, unfortunately, one cannot get the patient to even sleep. Probably the best drug as a sedative is hyoscyamin, one fiftieth of a grain hypodermically. This seems to be very reliable in the chorea of pregnancy in the moderately severe cases. In the worst case I have seen, nothing but deep surgical chloroform anæsthesia seemed to be of any use. Dr. Pollock's case was kept under chloroform for days at a time, by students relieving each other at stated times. And yet after this prolonged period she came out of the anæsthesia as bad as ever. She had to be fed with nutrient enemata, and she was allowed to come to sufficiently to swallow, and was then put under again. Of course she went from bad to worse, and one could not keep up this sort of thing for weeks and months on end. Dr. Pollock, thinking something pelvic was at the root of it—it was not known she was pregnant—asked me to see her. I found the uterus enlarged, the cervix patent; the breasts looked like gestation, and I concluded she was probably pregnant. I determined to try the so-called digital dilatation of the cervix. You are aware that Coleman, a Norwich doctor, originally described forcible digital dilatation of the cervix as one of the ways of curing the uncontrollable vomiting of pregnancy. He had seen a case, in consultation with a local doctor there, of uncontrollable vomiting of pregnancy, and he decided he must bring on premature labour, and he examined her, and as far as he could at the time he pushed his finger through the softened cervix and dilated it a little, and said he would come again in the afternoon to finish the dilatation. But when he came in the afternoon, to his astonishment, the vomiting

Digitized by Google

had absolutely ceased, and nothing further was done to that case. In subsequent cases he found in the considerable majority of instances of the vomiting of pregnancy, if he dilated the cervix the vomiting ceased. We know now that similar relief from the vomiting can be effected with less risk to the patient by applying some strong substance, such as iodine paint (1 in 4), to the cervix and endocervix. But I am speaking of this dilatation of the cervix, and Dr. Wade has described, in the Obstetrical Society's 'Transactions' for 1880, a case in which he thought that probably the same result might obtain in cases of chorea. He did it in a particular case, and the chorea ceased. Other cases have been reported of the same nature. So in this case I proceeded to dilate the cervix, getting my index finger through the cervix under chloroform anæsthesia, hoping not to bring on abortion. But I found, as soon as I got my finger in, that the fœtus was lying transversely, and that there were no membranes intervening. So that there was no object in delaying matters, and I was able to hook down a knee and deliver. The fœtus was already offensive, although there had not been much discharge. It had been retained *in utero*, and had been slowly degenerating, and toxins had probably been absorbed into the system, and may have caused the mental condition. The placenta was fatty and also rather unhealthy. As soon as the uterus was emptied the choreic movements abated, and her mind gradually cleared. Now comes an important clinical circumstance. I knew there was a tendency to septic trouble following these cases, and here we had a septic fœtus; and, of course, in detaching the placenta, I had left a very large raw surface where absorption could go on. I was therefore doubly careful, and we syringed out the uterus with 1-in-3000 mercurial douches, followed by borax douches. And yet the temperature rose on the first day to 101°, and on the third day to 104·5°, and then it gradually came down, reaching and remaining at normal on the fourteenth day. In six weeks she was discharged, but she had been semi-comatose and subsequently dull and stupid for almost the whole of those six weeks, an irregular form of puerperal insanity of the comatose type.

As regards the treatment again, in all such cases we must be particularly careful that there is nothing left in the uterus. In the treatment of chorea in pregnancy, the first thing is to try to get the patient to sleep, and to see that she is taking proper food. If it is a mild attack it will gradually wear itself out. We do not know sufficient about the poison to give an antidote yet, but we shall be able one of these days to detect the poison in the blood, I feel sure. Probably the best drug in the milder cases, where one simply wants a sedative, is the bromide, as given in this case. If sleep is wanting, chloral combined with bromide is necessary. In other cases, where these fail, one would try hyoscyamin, one-fiftieth of a grain. But in severe cases, if you find that drugs fail to act, or only act during their exhibition, you will have to empty the uterus, trying digital dilatation first. And as these severe cases are generally in the early months, I think the best plan would be to put the patient under an anæsthetic and clear the uterus out. At a later date one would feel inclined to dilate up the cervix and give the patient the chance of waiting twenty-four hours, to see if the chorea stops, if the membranes were found intact. If they were not intact, one would at once empty the uterus. This girl is already very much improved, and she will go on improving I have no doubt, although we may get a migration of the movements to the left side. It may for a little time be general, and then in another two or three weeks she will be quite well, for there is no albumin in the urine, and she sleeps and takes her food well, and the temperature is normal.

Note.—In the course of five weeks the choreic movements ceased, apparently under the influence of iodide of potassium, as prescribed by my house physician, Mr. H. P. Johnson.

Local Action of Suprarenal Extract on Nose and Throat (L. HARMER, 'Wiener Klin. Wochenschrift,' May 9th).—The reduction of the hyperemia that follows the local application of suprarenal extract to the mucosa of the nose and throat has a favourable effect in catarrhal conditions. It enhances the analgesic action of cocain, which property is probably due also to this vasoconstricting influence; possibly the cocain works more effectually on tissue free from blood. Suprarenal extract is, therefore, indicated in cases that require cocainization, as the amount of cocain can be materially reduced. This is the only positive indication announced as yet from Chiari's clinic in regard to the action of suprarenal extract on the nose and throat, after considerable experience.—*Journ. Amer. Med. Assoc.*, June 22nd.

Digitized by Google

ON THE TREATMENT OF FOUL WOUNDS.

A Clinical Lecture delivered at Guy's Hospital, June, 1901.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

PERHAPS few cases may make more demand upon the skill and judgment of a surgeon than does the treatment of what we may call for convenience sake a foul wound. How often is one tempted to close up an incised wound after having carefully cleansed it with germicides, and how disappointed and annoyed one is to find the part swollen and red, and inflamed later. This is particularly the case if deep and important structures, as tendons and nerves, have been divided, and whose edges have been brought into accurate coaptation by sutures.

In the case of foul wounds the proverb "More haste less speed" is particularly applicable as far as the early closure of the wound is concerned.

Perhaps I can best illustrate the methods which I believe can be adopted most advantageously by describing certain specific conditions.

Take, for instance, an ordinary compound fracture of the tibia and fibula produced by indirect violence, and presenting an aperture in the skin through which one of the sharp spike-like extremities of the tibia has been protruded. There is no laceration or bruising of the soft parts other than that consequent upon the passage of the fragments of bone in several directions.

In these compound fractures the treatment is accompanied with much greater difficulty and uncertainty than in the case of the simple variety. In the latter, a surgeon who is aseptic in his methods, who takes care never to introduce his fingers into the wound, who makes an incision sufficiently long to allow of the complete exposure of the fragments, and their free manipulation by his instruments—an incision often a foot in length in the case of fracture of the long bones of the leg—and who is familiar with the use of the screw and understands how to employ it, can guarantee almost with absolute certainty the perfect restoration of the anatomy of the part to that which existed previous to the injury. House surgeons at Guy's Hospital have been operating on these cases for me at intervals for several years, and they have effected their purpose most skilfully, and without risk to the patient's life.

Plate I is a typical example of a simple spiral fracture of the tibia and fibula treated in this way.

Such, I am sorry to say, has not been the experience of surgeons generally. Last year I placed before you in a lecture a statement of the operative measures I had adopted up to date. This was published in the 'Lancet,' May 26th, 1900.

Since this there was a discussion at the meeting of the British Medical Association on the treatment of simple fractures. It was opened by Mr. Bennett, who devoted a great deal of time and infinite pains to arrive at an effective working treatment. He dealt with the subject in his usual masterly manner, and I think he succeeded in proving his point not only to his own satisfaction, but to that of all those who took part in the debate. Unfortunately I was not present at the meeting, but have to depend on an abstract of the remarks of the several speakers in the report in the Journal ('British Medical Journal,' October 7th, 1900) for details as to the discussion upon the paper. I am sorry to see that there was this general consensus of opinion as the conclusion at which Mr. Bennett arrived after making inquiries from about three hundred surgeons in London, the Provinces, Ireland, and Scotland was that the operative treatment of simple fractures "excellent as it is in itself under certain circumstances, is not one that can be practised as an ideal plan of treatment by the general body of practitioners; it is in other words a method which is better restricted to those who are constantly in the habit of operating, and whose circumstances are such that the provisions for ensuring asepsis are as perfect as possible."

The evidence on which he relied is based chiefly on the results of the operative treatment of fracture of the patella, perhaps the very simplest of all such operations. It is one that offers no conceivable difficulty whatever to its perfect performance in the vast majority of cases, and in which the surgeon has no excuse for failure, since failure must, of necessity, mean some want of care on his part or of perfection in his methods.

I will give you the evidence he adduces in his own words. The answers are in response to one

Digitized by Google

of several questions addressed to the surgeons of Great Britain and Ireland.

"*Question* 7.—In cases of fracture of the patella treated by immediate suture, have you had any experience of results which are defective (*e.g.* with partial stiffness of the knee), or serious (*e.g.* terminating in amputation, in suppuration, or in complete stiffness at the knee)?

111 (one hundred and eleven) specific replies were received to this question, that is to say, from a large proportion of those surgeons giving information who operate in fracture of the patella.

Of these 7 have had no unfavourable cases themselves, and make no mention of such in the practice of others; 14 have had no unfavourable cases themselves, but *know of such* in the practice of others; 7 have had no unfavourable cases themselves, but *have seen such* in the practice of others; 3 have in their own experience had *suppuration* around knee, with impaired movement; 16 have in their own experience had *suppuration* and disorganisation of the joint, with *complete stiffness;* 8 have in their own experience had to *amputate;* 1 mentions suppuration, with *great danger to life;* 3 have had fatal results (one each); 2 make reference to (*a*) "More than one fatal case in the practice of others," and (*b*) "Certain fatal cases;" 12 have experienced *stiffness with material disability;* 3 have seen failure of aseptic precautions followed by "*serious results;*" 7 have seen stiffness without disability; 1 says the movements are never quite perfect; 5 have had experience of *re-fracture* or *cutting* through the bone by the wire during attempts at restoring movement, necessitating a second operation; 1 hesitates to answer the question without first consulting his solicitor. The remainder mention recurrent arthritis, necessitating removal of wire; results, "No better than in cases not submitted to operation," etc.; three surgeons have given up the operative treatment, and have reverted to non-operative measures."

Now I take it that these answers represent the *best side* of the case, as it is hardly to be expected that such a question would be answered fully and accurately by every one written to on the subject if his results had been equally unfavourable.

In the face of this evidence, and guided by the following principle, which he has laid down, viz. "The general value of a treatment cannot be gauged by the results which can be achieved by it in the hands of a few individuals, but must depend rather upon its adaptability to the circumstances and requirements of the general body of practitioners who use it," it is not surprising that he has arrived at the following conclusion.

"My opinion is that the treatment of fracture of the patella will in the future tend to resolve itself *with the majority of practitioners* into what I call the rational method; that is to say the early resort to passive movement upon the lines to which I have referred; and that operation, being reserved for the more severe cases, will become less common than it is now; because in many cases equally good results are obtainable without the risk that operation entails."

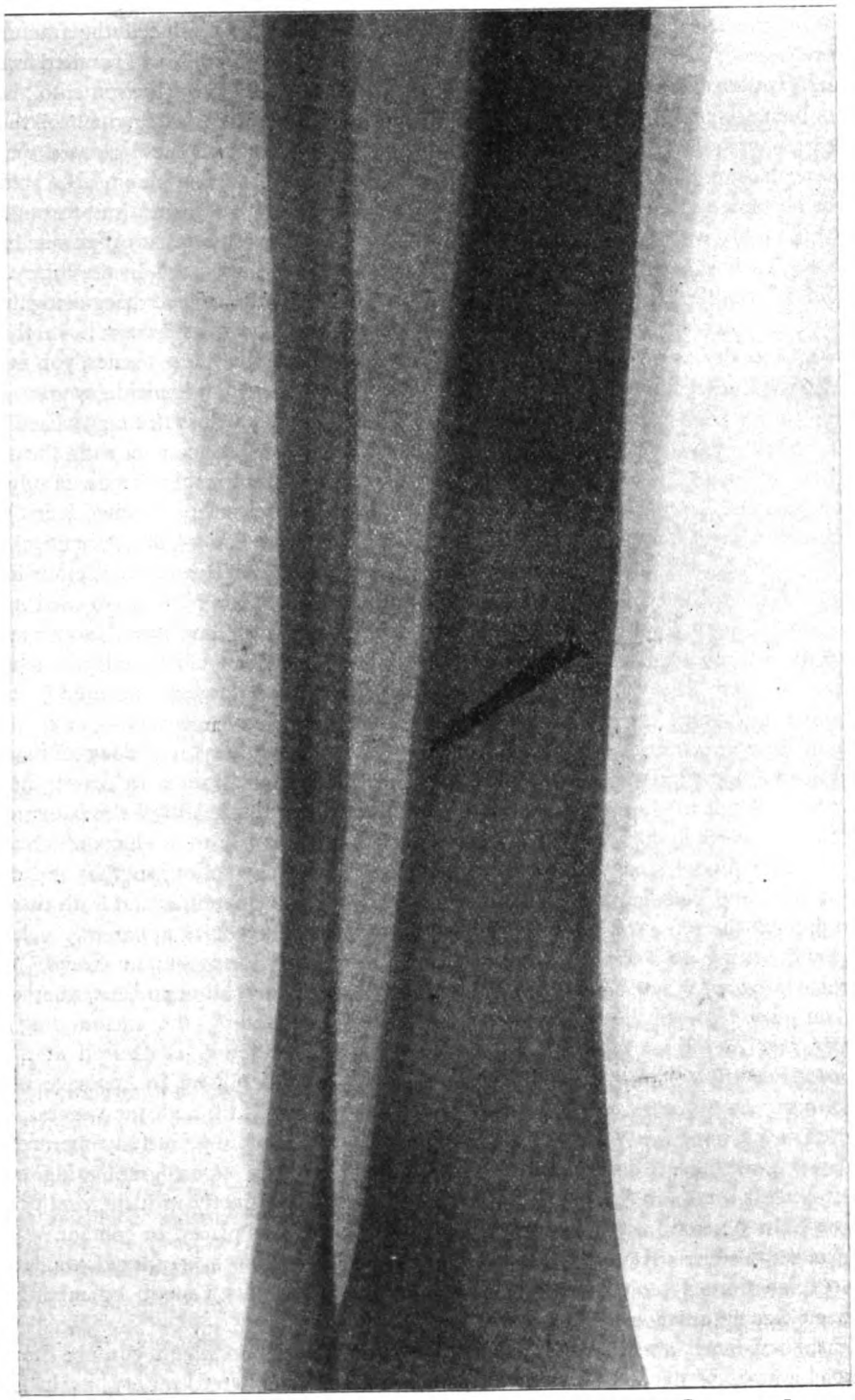
This seems to me to be a terrible conclusion to arrive at, since *it is nothing more or less than a charge of surgical inadequacy or insufficiency, whichever you like to call it.*

I object most strongly to this levelling down principle, and I do not for a moment believe that it will appeal to you. If surgeons are not sufficiently clean and skilful to perform such a simple operation as wiring the fragments of a broken patella without fouling their wound, it is hopeless to expect them to undertake successfully a spiral fracture of long bones, and especially those of the legs. No, gentlemen, we must level up and not down. If the principle of the treatment is avowedly recognised as right, and the methods necessary to effect it are considered unsatisfactory, let those methods be improved and do not let the treatment be watered down to meet the capacity of the average surgeon. The same applies to every difficult operation in surgery, and such an avowal at such a meeting at this period of the world's history is, I say again, appalling. I would urge on you all to read Mr. Bennett's most judicious and clearly stated address and the discussion upon it as published in the 'British Medical Journal.' The medical profession at large owes a deep debt of gratitude to Mr. Bennett for his most valuable contribution to surgery.

Returning to the consideration of the compound fracture, from which I have momentarily digressed, you should without delay put the patient under an anæsthethic and thoroughly clean the skin of the limb. In the case of the tibia and fibula, the foot and leg should be treated with plenty of soft soap,

Digitized by Google

PLATE I.—Spiral fracture of tibia treated by screwing.



Digitized by Google

lysol, and other germicidal lotions, up beyond the knee, till all the loose hard skin has been effectually removed. Then an incision is carried upwards and downwards from the aperture in the skin till the fragments have been exposed sufficiently to permit of their being properly manipulated, the muscles and soft parts being separated from them. Foreign material is carefully looked for and removed, together with any lacerated bits of muscle. The surface of the wound and the ends of the bones are washed thoroughly with some efficient germicide, and when this has been done, the parts are carefully dried. Cyanide gauze, which has been sterilised in 1 in 20 carbolic lotion, and has afterwards been wrung as dry as possible out of boiling water, is packed into every crevice of the wound, and the limb is surrounded with the same material. This is removed at the end of twenty-four to thirty-six hours, when the wound is perfectly dry. The bones can under these circumstances be screwed or wired together with the greatest ease, as compared with the difficulty which is experienced in a simple fracture. This facility of effecting apposition in the case of the compound fracture is due to the escape of blood through the wound, as opposed to its extravasation into the soft parts, with the consequent shortening up of these inextensile ties of the bones in their length in the simple fracture.

After the operation, if any hæmorrhage takes place, it may be wise not to close the wound completely, but to pack partially for one or two days. Even with all these precautions, you may not be able to free the wound sufficiently completely of organisms to prevent the screw or wire acting as a foreign body and setting up sufficient irritation to require its removal *after it has done its work* and union has taken place between the fragments. *You must remember that to leave a piece of metal in a wound necessitates a much higher degree of cleanliness, or in other words, perfection of asepsis, than is requisite to close such a wound without it.*

I notice that several surgeons have ascribed their inability to keep their screws in the wound to their not being perfectly silvered, and have suggested screws made of solid silver. I need not say that this not the explanation of their failure. Except in a very few solitary cases, as you know, we have used the ordinary carpenters' screw, which, I believe, is supposed to be made of steel.

Now, supposing the compound fracture has resulted from direct violence, the condition of the soft parts is often very different.

The skin, muscles, and fat may be mashed up by the force which produced the fracture, and more fouling of the wound have resulted from the clothes or dirt, or both, being driven into the wound. In such cases the free laying open of the wound, the careful washing of the lacerated parts, and the packing of the wound may not be sufficient to prevent the spread of organisms through the tissues, whose vitality and resisting power have been reduced to the lowest ebb by the injury. If you feel that this is probable, and recognising that any spread of infection will rapidly result in the death and decomposition of these tissues, you must introduce into the wound a germicide whose action is progressive, and which is not neutralised within a very short period by its contact with the tissues. The germicide I use for this purpose is sulphur. Several years have now elapsed since I first published in the 'Medical Week' of December 9th, 1893, a paper entitled, "How far is sulphur likely to be of use to the surgeon?" I have used it continuously since then, and have saved very many lives and limbs which I should have lost without it. It has also been used largely and equally successfully by others.

I might remind you of a few of its qualities:

(*a*) Sulphur appears to exert no deleterious influence on the health of the patient.

(*b*) It gives rise to products which are powerfully caustic in their action, so that the drug must be used in small quantities and with discretion. The most active agent is apparently sulphurous acid, which rapidly forms sulphuric acid.

(*c*) It destroys all organisms, whether in a wound cavity or invading the surrounding tissues. Its germicidal function is exerted at a considerable distance, probably by the passage of the nascent sulphurous acid through the tissues.

(*d*) It acts more powerfully upon recently-incised structures than upon granulating surfaces. Its action varies directly with the vitality of the tissues with which it is placed in contact.

(*e*) Its action is rendered more uniform and general and less violent by mixing it with glycerine.

The manner in which you use the sulphur is to use the precipitated variety, as it is much finer than the sublimated. It is mixed in a paste with

Digitized by Google

glycerine, and a piece of sterile gauze is thoroughly permeated with it. As soon as the wound has been dried, this gauze is packed into every crevice till it is completely full. Over this a large hot boric acid compress is placed, and it is changed every two or three hours. By looking at the wound, and especially by raising a portion of the gauze from the raw surface, you can readily determine by the amount of slough the length of time the sulphur should be allowed to remain in it. As I said, this period varies inversely with the vitality; the greater this is, the less the period requisite for effectually sterilising the wound. This period varies between twenty-four hours and several days. Indeed, if the tissues are very much lacerated, it may be used for many days without fear. When the sulphur has produced a sufficient slough it is removed, and the wound is packed with cyanide gauze, sterilised in carbolic lotion, and wrung out of boiling water. The compresses are continued till the slough has separated, and the wound is perfectly red and clean throughout. The fragments are then brought accurately together, and are retained immovably in this position by a stout wire or screw, as may seem advisable. As there is always a fair probability of the process of sterilisation not being complete, accurate notes should be kept of the locality and direction of the screw or wire, so that it may be easily removed when it has ceased to be of use should it serve as a source of irritation.

The Employers' Liability Act will render such conservative treatment much more usual in the future, since the patient and his family are kept in comfort during his convalescence, and time is a matter of comparatively little moment to him.

Some Observations on Chronic Seminal Vesiculitis.—Arthur L. Chute and Richard F. O'Neil found sixty cases of this affection among 540 patients presenting various sorts of genito-urinary disease. The treatment which has given them the best results is massage of the vesicles and ducts, and of the prostate *per rectum*. At first the time of massage should be very short, and at intervals of from five days to a week. Later the intervals are longer, and the massage is more thoroughly done. In some cases of long duration, dilating with Oberlaender's or Kollmann's dilator seems to have a beneficial effect.—*Medical Record*, June 22nd.

PRESENT DAY SKIAGRAPHY.

WITH DR. HUGH WALSHAM AT ST. BARTHOLOMEW'S HOSPITAL.

DEMONSTRATION IV.

GENTLEMEN,—In cases of cardiac hypertrophy and dilatation, the X rays will show the amount of hypertrophy or dilatation, as the case may be, better perhaps than by the ordinary means of percussion. The following picture is one from an enlarged heart of chronic Bright's disease. It shows very well the displacement outwards and downwards of the heart's apex, and also some

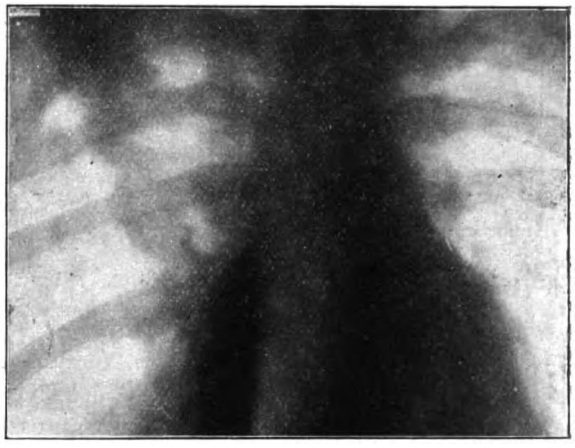


FIG. 1.—Hypertrophied and dilated heart of chronic interstitial nephritis.

commencing dilatation in addition. The next picture is the heart of a woman with well-marked mitral stenosis. The heart in this disease takes a very peculiar shape, in fact, cases of mitral stenosis might be picked out from other cases of morbus cordis with the screen. It may be described as purse-shaped, this form of the heart being produced by the hypertrophy and dilatation of the right side over and above that of the left. And while I am on the subject of mitral stenosis, I may say that I think the X rays may help us in some of those cases of mitral stenosis in which we suspect tubercle.

It is a well-known clinical and pathological fact that mitral stenosis is almost in itself a bar to the occurrence of tubercle in the lung. Such cases undoubtedly do occur, but very seldom. When a case of mitral stenosis comes before us, for instance,

Digitized by Google

and we find crepitations in some portion of the pulmonary area, we must not, I think, put the same amount of stress upon this sign as we otherwise should be justified in doing were the mitral stenosis absent. In fact, in some of these cases it is extremely difficult to say from observation with

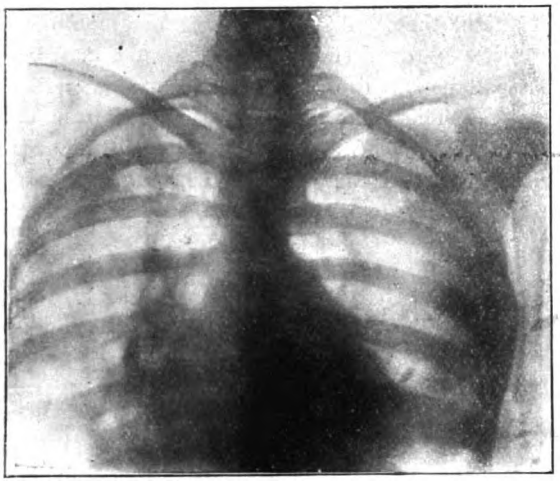


FIG. 2.—Mitral stenosis.

the stethoscope only whether the lung be affected by the tubercle or not. Here the X rays come to our aid and show whether the lung is so affected. Whether it will be ever possible to show valvular disease of the heart is extremely doubtful All my attempts up to the present time within the body have been complete failures. Outside the body the calcareous valve shows distinctly enough, as the following picture well exemplifies. Calcareous blood-vessels and atheromatous patches in the aorta probably throw such indistinct shadows that nothing can be made out from them. Another interesting point in connection with X-ray work in cardiac disease is the systole and diastole of the heart, as seen on the fluorescent screen (see 'Lancet,' May 18th, 1901). The systole and diastole of the heart is clearly seen, and in some cases, especially in thin persons, the systole and diastole of the auricles can be made out. I have made many attempts in patients with reduplicated second sounds to determine if one could detect any difference between the systole of the two ventricles; but I must admit that up to the present I have not been able to differentiate the systole of the one from that of the other.

The next medical disease to which I will pass on is that of renal calculus. Some radiographers maintain that all calculi of a certain chemical composition can be detected by the Röntgen rays. In fact, some would go so far as to say that in a case of suspected renal calculus, if the patient has been examined by the X rays and the result be a negative one, the opinion can be confidently expressed that a renal stone is not present. With these observers I cannot agree. To give my own experience, we will take twelve consecutive cases that were X-rayed in the electrical department of St. Bartholomew's Hospital for suspected stone in the kidney. In three out of the twelve I could say definitely that a stone was present. In the rest I could give no opinion one way or the other, the negative being perfectly blank, as far as the renal region was concerned at any rate. Yet in one of these cases a large calculus was subsequently removed by Mr. W. J. Walsham, the surgeon in charge of the case. Again, for renal calculus to be detected by the X rays, the stone must be of a certain size. Probably stones which are smaller than a good-sized pea will escape detection alto-

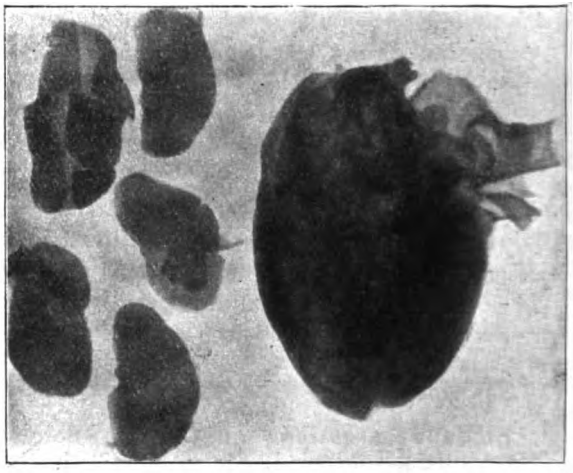


FIG. 3.—Heart with calcareous mitral valve, with spleen and kidneys from the same case; also kidneys from another case of mitral disease. The kidneys and spleen contained infarcts.

gether by an X-ray examination. This is very well illustrated in the following case. A woman thirty-three years of age was admitted to St. Bartholomew's Hospital under the care of Mr. Butlin. The diagnosis was a doubtful one, halting between a tuberculous kidney and a renal stone. The

Digitized by Google

patient was sent to the electrical department for an X-ray examination, and the following skiagram (Fig. 4) is a picture of this case. It is clearly seen that two, if not three, large stones are present. On this diagnosis Mr. Butlin removed the kidney. is the skiagram of the kidney. It will be seen that the three larger stones shown in the skiagram taken during life are perfectly evident; but on looking carefully at the picture it will be seen that there are a considerable number of other small calculi,

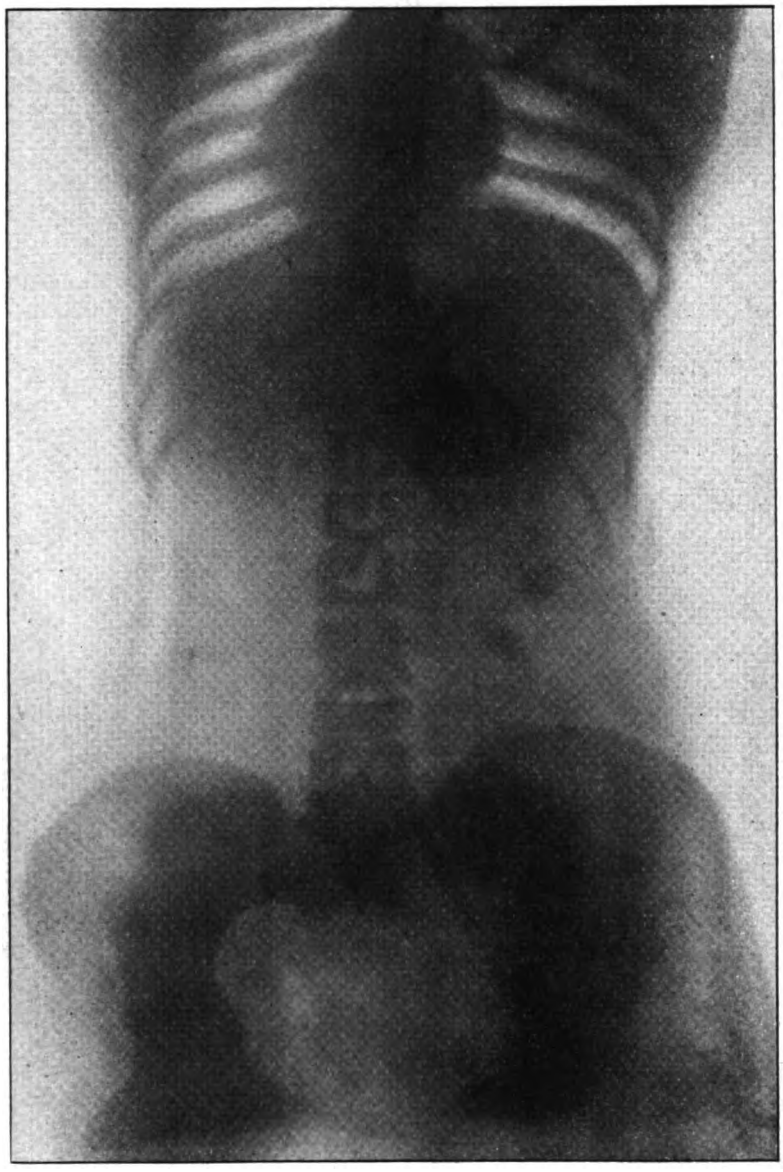


FIG. 4.—Calculus in the right kidney. [This Fig. and also Figs. 5, 6, and 7, are by permission from the 'Archives of the Roentgen Ray,' published by Rebman, Limited.]

The organ was sent to the electrical department immediately after removal, and was put on a photographic plate and exposed to the rays for a period of twenty seconds. The following picture (Fig. 5) varying from the size of a large pea down to a mere sandy deposit. Now I think this case makes it perfectly clear that only stones of a certain size can be shown by the rays, and to say that

Digitized by Google

because no shadow of a stone is shown on the skiagram that therefore no stone is present, is going further than our present methods of examination justify us. I will now give another case of stone in the kidney, and with that I must be content.

A woman, thirty-three years of age, was admitted to St. Bartholomew's Hospital, under the care of Mr. Butlin. Here the diagnosis was almost certain. A large number of small calculi had been passed by the patient during the three previous years. On taking a skiagram (Fig. 6) of this patient a large stone, or apparently two large stones were seen in the left kidney. The kidney was removed, and, as in the former case, it was sent to the electrical department, where it was at once skiagraphed (Fig. 7). On looking at the plate apparently two stones are present, but when the organ was cut open and dissected up I found that there was only one stone. The appearance of two stones being present was due to the way in which the stone was moulded to the pelvis of the kidney.

Now with reference to the chemical composition of renal calculi, I am perfectly certain that this must be taken into account in forming any opinion as to whether a stone be present or not. Some pure uric acid stones are comparatively transparent to the rays. Next in order of opacity come the phosphatic stones, and finally the stones most opaque to the rays are the oxalate. The chemical composition of the stones in the pictures above given was a mixture of oxalates and phosphates.

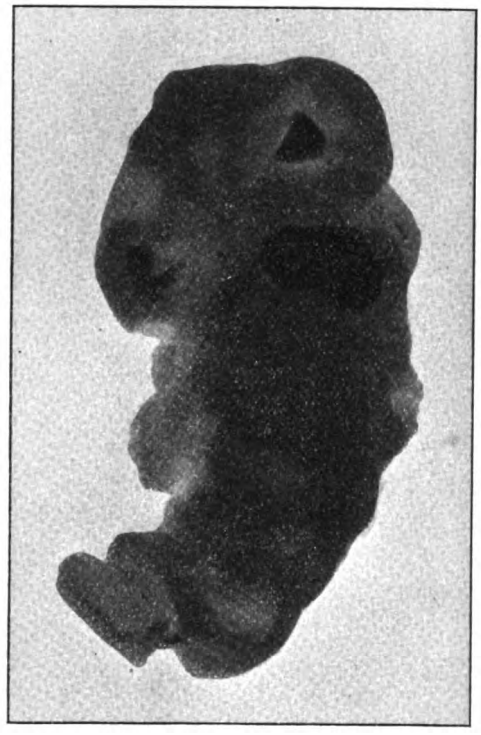


Fig. 5.—Calculus in the right kidney.

Now a word with reference to the time of exposure. Of course in attempting to skiagraph an abdominal organ the time of exposure should be as short as possible, for this reason, that the rise and fall of the diaphragm, and the rise and fall of the abdominal wall in respiration is liable to greatly obscure the shadow of the stone. So, if possible, in taking a skiagram for a suspected

Digitized by Google

renal calculus the plate should be as rapid as possible, and the exposure should be as short as possible. If it can be reduced to the time in which the patient can take a deep breath and hold it while the operation is going on, so much the more likely will you be able to produce a definite sharp shadow, supposing a stone be present. Stones impacted in the ureter, of course, can be shown as easily as when impacted in the kidney. The only other medical stone, if I may use such a term, in which the X-rays are occasionally applied for the purpose of diagnosis is in the case of gall-stones. And here, again, gall-stones vary, outside the body at any rate, in their opacity to the rays, according to their chemical composition. Probably all pure cholesterin stones

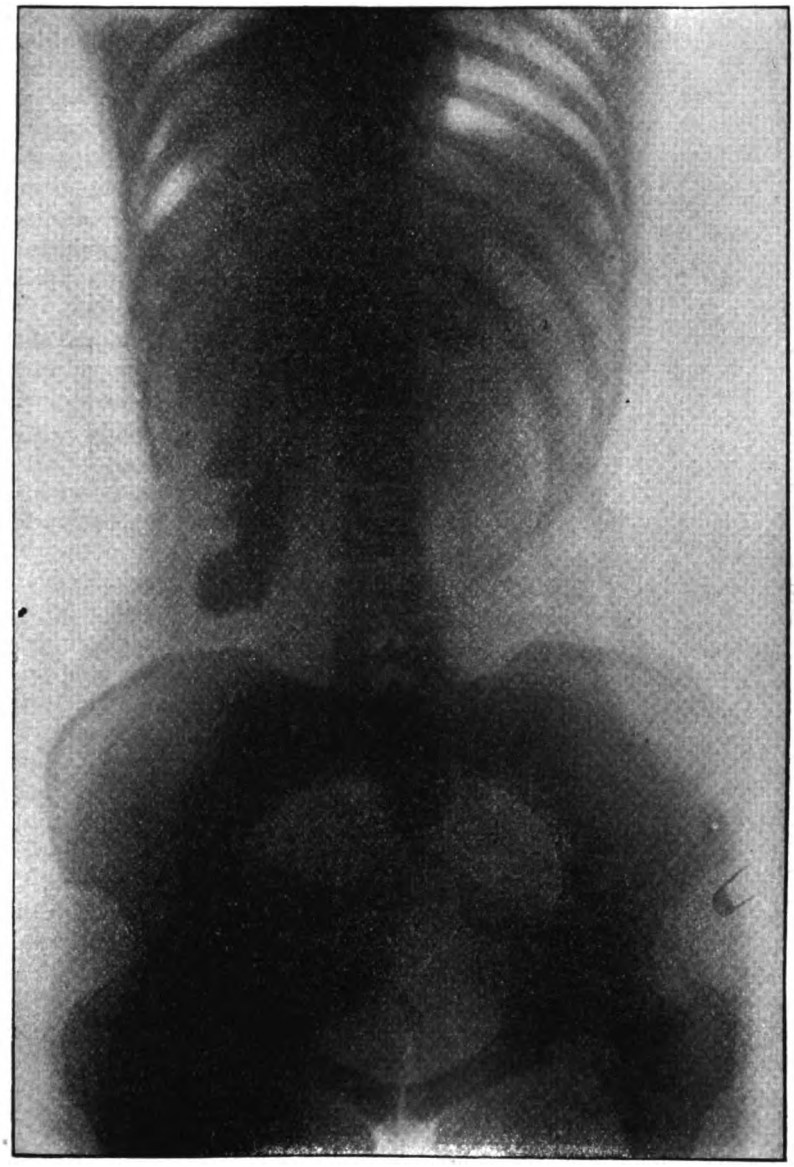


FIG. 6.—Calculus in the left kidney.

Digitized by Google

or the mixed cholesterin and pigment stones are almost transparent. The calcium phosphate stones offer a much greater resistance to the rays, but they are very indistinct. I have tried three or four times to obtain skiagrams of gall-stones within

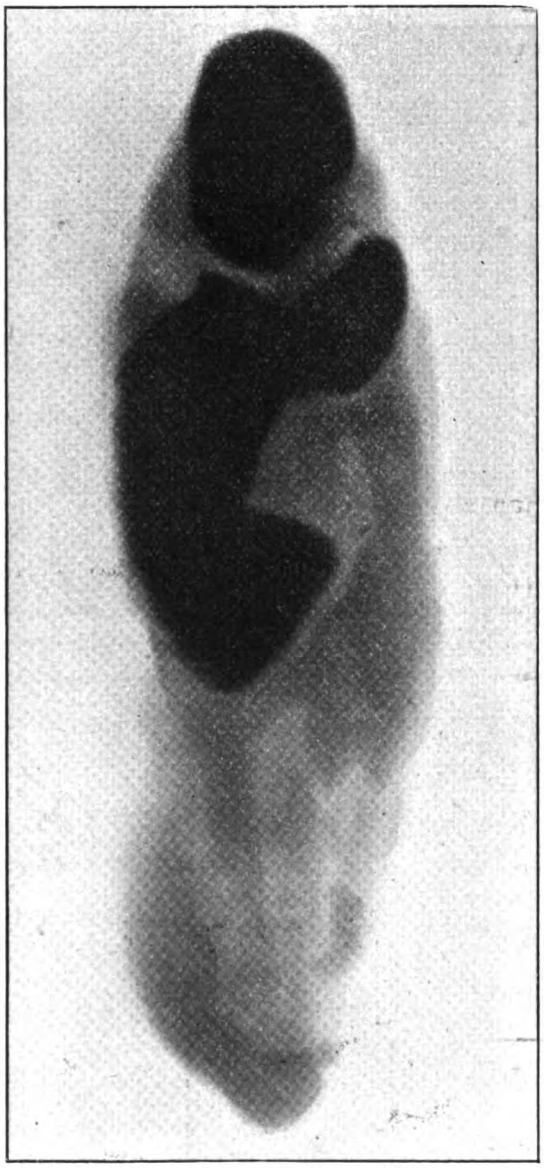


FIG. 7.—Calculus in the left kidney,

the gall-bladder which were known to be present, but each time I have met with nothing but failure. In a case of a large gall-stone impacted in the intestine, of which I tried to get a picture, I again met with nothing but disappointment. This stone was as large as a small hen's egg.

And here, gentlemen, I must stop. I have in these demonstrations endeavoured to place before you, as clearly as I could, the uses of the Röntgen rays in clinical diagnosis of thoracic and abdominal disease.

"Neglected, but Valuable, Therapeutic Measures," was the title of a paper read by Geo. F. Butler, at the fifty-second meeting of the American Medical Association. After reviewing a number of methods and remedies which were popular before the scientific era in medicine, many of which were deservedly abandoned, *e.g.* scatological remedies, animal products, etc., he directed attention to some measures which should not be neglected by the profession. Prominent among these is blood-letting in a class of cases in which its powerful physiological properties would be desired. Among these he cited cases of toxæmia, in which the depleted blood could be substituted by normal salt solution. Counter-irritation is a neglected, but a valuable therapeutic resource, in illustration of which he directed attention to the remarkable change for the better in the condition of some cases of insanity after accidental fracture of a bone. Bathing, used in typhoid fever with great advantage, is also very valuable in yellow fever. The combination of drugs in a single prescription has fallen into undeserved disrepute as polypharmacy; but he claimed that very often a combination of remedies so modified their action that a new and superior therapeutic agent was obtained, for instance, the combination of iron with the simple bitters in the treatment of chlorosis, especially for overcoming anæmia in children. The infusion of quassia combined with aromatic sulphuric acid and tincture of iron, he is accustomed to give with great advantage for this condition. The sexual erethism seen in the condition of auto-toxism is relieved by (1) removal of toxic agents by purgatives, and (2) by heart tonics. A valuable but neglected agent for the latter purpose is copper sulphate and copper arseniate, given in small doses. Massage is also a measure not sufficiently appreciated. He especially recommended a combination of massage, hydropathy, and out-door exercises as having great restorative value. Suggestive therapeutics is worthy of more study as a potent agent for good, and of great value when used in conjunction with other measures just referred to.—*Philadelphia Medical Journal*, June 22nd.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 456. WEDNESDAY, JULY 24, 1901. Vol. XVIII. No. 14.

CONTENTS.

PAGE

*A Clinical Lecture on the Diagnosis, Prognosis, and Treatment of Quiescent Tuberculosis. By J. MITCHELL BRUCE, M.A., LL.D., M.D., F.R.C.P. ... 209

*A Clinical Demonstration at the Medical Graduate's College and Polyclinic. By C. O. HAWTHORNE, M.D., M.R.C.P. 214

*Arsenical Poisoning from Beer-drinking. By T. N. KELYNACK, M.D., M.R.C.P. 222

Bacteriology of the Urine. LINDLEY SCOTT, M.D. 224

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

THE DIAGNOSIS, PROGNOSIS, AND TREATMENT OF QUIESCENT TUBERCULOSIS.

Delivered at the Brompton Hospital for Consumption, May 22nd, 1901.

By J. MITCHELL BRUCE, M.A., LL.D., M.D., F.R.C.P.Lond.,
Consulting Physician to the Hospital.

GENTLEMEN,—My first word to you to-day is one of apology for the title of this lecture. The subject of it, as you know, is quiescent tuberculosis in relation to its diagnosis, prognosis, and treatment. This is far too large a subject for one to take up and dispose of in a short hour, and so the title is misleading, for some of you may have expected that I would cover far more ground than I propose to attempt to do. Perhaps you may consider that I ought to apologise for the selection of the subject at all, inasmuch as quiescent tuberculosis may appear to you to be a subject of no particular interest. It is tuberculosis in the condition of arrest or repair, and some of my audience may ask if we have got our patient well after a great deal of trouble, why not leave him alone, and turn our attention to active tuberculosis instead of quiescent, arrested, healed tuberculosis. Well, I hope to show you presently there are some good reasons why, after all, quiescent tuberculosis is worthy of careful consideration.

And first I would remind you by way of introduction that this quiescent tuberculosis of which but little notice is taken is far more common than active tuberculosis. How very common arrested phthisis is may be gathered from post-mortem facts to begin with. According to authorities upon whom we can depend both in this country and abroad, somewhere between 30 and 40 per cent. of all dead bodies of persons between ten and forty years of age opened contain evidence of tuberculosis—an enormous percentage: and unquestionably a very considerable proportion of these must

Digitized by Google

be instances of quiescent tuberculosis. Then there is that striking statement of fact, a paradox and a startling one, that tuberculosis is the most curable of all diseases. I believe that it is perfectly correct, and it is in accord with what I have just said, that there is an immense number of people in our midst, apparently well, who are the subjects of quiescent tuberculosis. Now I am sure all of you here will agree with me, for you are qualified men, and men of knowledge and experience, that quiescent tuberculosis is not dead but dormant tuberculosis. The bacilli in arrested tuberculosis are virulent and capable of growth and reproduction under circumstances favourable to them. It matters not whether the disease be in the lungs or in the pleura, or in the peritoneum, or where it may be, quiescent tuberculosis constitutes a standing danger to health and life. Now we begin to see the extreme importance of the subject. Tuberculosis is a disease which when it is apparently past and gone is not really so, for the time and opportunity may come when it will wake into renewed activity. Let us consider this subject then from the practitioner's point of view, and let us this afternoon deal with only a certain number of directly practical points, not with the whole broad question of dormant or arrested tuberculosis.

The Pathology of Quiescent Tuberculosis.

First of all, however, it would be well for me to remind you of some facts relating to the pathological anatomy of this condition. We must always base our ideas on pathological facts, if we possess them. What are the pathological facts? Suppose we take the lung. What is the condition we find in the lung which we comprehensively name arrested pulmonary tuberculosis? Tuberculosis may become arrested at different periods of its evolution. Each individual miliary tubercle may become hard and obsolete, when it remains as a tiny horny grey nodule. In the next place, if we take an area of lung infiltrated with tubercle, the pneumonic consolidation which usually accompanies and follows this infiltration may become fibroid; and the involved areas, as you know, may be of different sizes. This is the second form in which we find quiescent tubercle. In the third place, such an area may caseate, and as the result of this caseation we may have an ordinary cavity which dries up and shrinks more or less, in some instances remaining perfectly quiet. Again we know that tuberculosis of serous membranes is very commonly arrested. First there occurs an exudation or an effusion which disappears or is removed, to be followed by adhesions and condensation of the neighbouring tissues. Fifthly, you may have solid organs like the lymphatic glands, kidneys, and testes caseating in areas which remain encapsulated. Now in all these five conditions, at least, the disease may for the time being be arrested. This is but a very bald description of individual lesions and modes of repair. Think for a moment of the condition of the lung in ordinary quiescent pulmonary phthisis after a sharp attack of tuberculosis; several of these different changes are associated together in the most complex manner, and in addition to them there are other morbid states, including emphysema, bronchial catarrh, secondary cardiac changes, blood changes, and so on. Let us turn for a moment to the bacteriology of arrested tuberculosis. On microscopic examination of any of the five conditions mentioned above, we know that we may find not only evidence of dead bacilli but also evidence of living bacilli. The bacilli will cease to fix the colouring reagent, but though they do not take on the stain, they are, as I have said, virulent, and they can reproduce themselves, that is, they are infective. Thus you see while we have several gross pathological changes which would be regarded as repair and which are indeed of the nature of repair, yet the bacilli still possess potential force which will break out again into renewed activity when the patient's condition is sufficiently depressed. So much for the pathological conditions.

Clinical Characters.

Now let us review very briefly the ordinary clinical characters of quiescent tuberculosis. The subject of this disease is healthy, though unsound; he is variably disabled according to the organ involved, and the extent and depth of the pathological changes; he may be delicate, anæmic, or even cachetic in appearance, but more often, if all cases are taken into account, he is not; sometimes he is thin, but possibly he is well-nourished, or even

Digitized by Google

stout. If the case is one of arrested tuberculosis of the lungs, there is, as a rule, some cough, especially after sleep, and therewith expectoration and breathlessness on exertion, and a little palpitation of the heart. The body temperature is not above normal. Digestion and assimilation are natural and active. The man is able to do a fair amount of work with success and enjoyment, but as a rule he is glad when it is over for the day. This state of comparative health and strength is occasionally interrupted by attacks of catarrh, with increase of cough and expectoration, and possibly a little fever with its ordinary phenomena and effects; or hæmoptysis comes, causes temporary alarm, and goes. The physical signs in the lungs are naturally as varied, and possibly as complex as the lesions I have reviewed, in their seat, extent, characters, and course. Just as the leading changes in the repaired lungs are fibrosis, induration, adhesion and shrinkage, so the principal physical signs mainly are local flattening of the chest-walls, depressing of the shoulder and tilting of the scapula, diminished movement, impaired resonance on percussion, weak respiratory sounds of bronchial character, and weak small crepitus in inspiration, or with cough. Occasionally there are signs of a cavity, which may be either dry or moist. Very often the familiar signs of emphysema and bronchial catarrh are prominent—possibly to such an extent as to be misleading.

Now I can understand that you may say that all this is very elementary description, and that nothing is so easy clinically as to trace the progress of repair and arrest of tuberculous lesions by physical examination. I entirely agree with you, and I apologise to you for having occupied so much of your time over it as I have done. But let me say that I have submitted to you this simple or easy aspect of the diagnosis of quiescent tuberculosis in the chest with the deliberate purpose of bringing home to you its difficulties. My opinion is that we are constantly committing the mistake of failing to diagnose quiescent pulmonary tuberculosis. Often we miss it altogether; more often we discover it, but regard it as active tuberculosis; often, also, we fail to appreciate its true relations to other pulmonary conditions of the first importance. And, having erred in our diagnosis, we are led astray in prognosis and treatment. Let me illustrate my contention by a few cases.

Hæmoptysis in Quiescent Tuberculosis.

A man of forty is taken with violent hæmoptysis, which recurs every one or two days for ten days, and when physical examination is permissible the left apex is found to be dull and crepitant. He is diagnosed to have active phthisis, is advised to continue to rest from work for months, and seriously contemplates giving up work altogether and emigrating to a better climate. However, when the man's case is more deliberately investigated, there are found the signs of fibroid disease of the left apex, and it is discovered that fifteen years before he had right pleurisy with effusion and was tapped, that five years later he had left pleurisy with effusion and was tapped, and that since the later date, although quite well and very active and industrious, he has had constant purulent expectoration. Now I wish you to particularly note the bearing of the diagnosis on the prognosis and treatment of this case, according as it is on the one hand correct or on the other hand incorrect. If the condition is interpreted to be one of early active tuberculosis, the usual uncertain hesitating prognosis has to be offered. Who can say what will be the result when the apex is found full of moist sounds and impaired in resonance at the end of a hæmoptysis in a lung previously sound? No one. The prognosis of the disease cannot be foreseen for several weeks at least, and so the case has to be regarded seriously, and all possible measures are employed to promote arrest, including abandonment of work, open-air treatment, and perhaps immediate expatriation. On the other hand, if the correct diagnosis be reached, *i. e.* that the hæmorrhage proceeded from an old lesion, that it was a casual occurrence, that there is no activity in the affected area, but only signs of fibrosis, *i. e.* of repair, the whole aspect and prospect and line of treatment are different. The very chronicity is a proof of the man's recuperative power. Now he may be assured that he will recover. He is told that in a few weeks he may resume work, and that he need not think of emigration. You may say that this is an exceptional case, but I can assure you my experience tells me precisely the contrary. I believe such cases are quite common. I am not speaking of hæmoptysis simply as a complication of ordinary chronic phthisis, but of hæmoptysis in the course

Digitized by Google

of pulmonary phthisis so thoroughly quiescent as to have been forgotten by the patient, and to pass unappreciated by the doctor. I will even go so far as to say that a very considerable number of cases of so-called "early hæmoptysis" or "incipient tuberculosis with hæmoptysis" are nothing of the kind, but merely instances of arrested phthisis, with fresh hæmoptysis as a passing event in their course. But there is another practical aspect in the hæmoptysis which has to be regarded. Just as hæmoptysis, followed by the discovery of a pulmonary lesion, often causes unnecessary anxiety, so if no lesion be found after hæmoptysis the occurrence is disregarded and forgotten. Now this conclusion is just as incorrect as the other. Hæmoptysis is never to be made light of. If it come and go, and leave no trace behind, the proper diagnosis is that it proceeded from a tuberculous focus that we have failed to find, and that the patient is the subject of quiescent tuberculosis. Some of the most furious hæmorrhages from the lungs are found to be associated with physical signs of fibrosis of very limited area, and in a situation where they might have been readily missed.

Association of Active and Quiescent Tuberculosis.

Now let me give you another illustrative case. A man of twenty-four was sent to me recently for severe cough of a paroxysmal nature, coming on particularly after food, and with what were called indefinite physical signs. It was added that he was neither wasting nor suffering from perspirations, but that he had done so recently. The question put to me was what could be done for the chronic paroxysmal cough? I found the signs of limited tuberculosis of quite recent development in the right apex, and in the left apex well-marked dulness and thick creaking adventitious sounds, whilst the apex of the heart was displaced outwards and to the left, and a patch of dulness or impaired resonance could be made out at the posterior base. This aroused my suspicion that the patient was the subject really not only of recent but of remote tuberculosis. And I found on enquiry what had apparently escaped the attention of his medical men, that as a boy the patient had suffered from pleurisy and what was called "a touch on the lungs" fourteen years ago, whilst he had slight cough for five years, although he was working steadily at his profession, and had recently undertaken further responsibility in connection with it. The bearing of the facts on the correct diagnosis of this case is obvious, and the bearing of the diagnosis on correct prognosis and treatment equally so. It is true that in this instance there is activity of the tuberculous process, but the recrudescence of activity is a very different matter from a primary outbreak. The man has been tuberculous for years, not for a few months, and the prospect of recovery is good, and the method to be adopted to promote recovery is far less severe than it would have been otherwise. One of the most valuable elements of prognosis in tuberculosis, and one for which search is always to be carefully made, is the history of previous recovery.

Pleurisy and Quiescent Tuberculosis.

I come next to a form of quiescent tuberculosis which introduces us to a series of prognostic and therapeutic considerations of the very first order. Take the following case. A man twenty-nine years of age is brought to me by his medical attendant for poor health and signs of tuberculosis in the right upper lobe. The doctor has just discovered the pulmonary lesion and is greatly disturbed. I find the physical signs—impaired resonance, blowing breath-sounds, and post-tussic crepitation—over the right upper lobe, both back and front, but I also find scattered dulness and crepitus to the base. On going into the man's history carefully, we make out that four years before he had pleurisy with effusion; that he went to an unhealthy locality to take up work; that after another four years, that is some six months before I saw him, he had hæmoptysis and a return of the pleurisy; and that he had not been strong since although by no means ill. It turns out you see that all the signs on the right side of the chest are old; that there is no active tuberculosis; and that the patient has been tuberculous for five years. Our anxiety is relieved.

Now you will observe that in this case I date the tuberculosis from an attack of pleurisy with effusion. You will have observed also that in the first case I narrated there was first one and then

Digitized by Google

another attack of pleurisy with effusion, both treated with aspiration. Here is the point on which I wish to insist: that quiescent tuberculosis often follows on pleurisy of what is known as "the common type." This is a consideration of supreme importance prognostically and therapeutically. It is now very widely recognised, but it is very slow in its influence on the treatment or management of early tuberculosis. The larger proportion of serous pleurisies are tuberculous, and after the disappearance or removal of the fluid from the chest the subject of the disease remains tuberculous, ready to suffer from pulmonary invasion. The common sequence is as in the cases I have just related to you: hæmoptysis occurs in one, two, three, or more years, and is very likely to be disregarded; then a fresh pleurisy occurs, whether serous or dry, and this too may be disregarded: then an apex becomes infiltrated, and now the cry is raised that the man has tuberculosis, and he is hurried into a sanatorium or out of the country. You will see, I trust, how incorrect throughout have been the diagnosis, prognosis, and treatment here.

Quiescent Tuberculosis of Serous Cavities and Glands.

So far I have referred to cases of tuberculosis within the chest only. If we extend our observations we find a similar order of events in other parts of the body. I have under my care at this moment in Charing Cross Hospital two cases of recrudescent tuberculosis of the peritoneum: two boys with return of tuberculous peritonitis, several years after successful treatment in hospital. And you know how frequently we find an old pulmonary lesion along with tuberculous peritonitis. Manifestly, we can never afford to make light of tuberculosis, however gratified we may be with what we call recovery from it or the cure of it. Unless we anticipate its recurrence and provide against it as far as possible, a fresh outbreak may only too readily be the result. By way of further bringing home this practical conclusion to your minds, I might narrate to you a case in which the primary manifestation of tuberculosis was epididymitis. The second manifestation pulmonary in its seat; or an instance in which first the ear, then the bones, and then the apex of one lung were attacked in succession, but the lung so long after the other organs that the person was regarded as perfectly sound And so with glandular tuberculosis and other forms of the disease.

Conclusion.

I need not insist on my position any further. I trust I have made it clear to you that although we all acknowledge in the abstract that tuberculosis is extremely common, affecting a considerable portion of the community; that it becomes arrested in a large number of instances, whether spontaneously or by art, and that it breaks out again in the same or in a different form when the vitality of the subject of it is lowered; yet, when we meet with an instance of such outbreak we do often fail to grasp the situation, and to prognose and treat correctly. How is this error to be avoided? First, and chiefly, by obtaining a careful history of the case. It is because we do not take the trouble to do this that we fail so often in our diagnosis. We assume that the present is the first event. And what is more, you must remember that tuberculous patients and their relatives often lead us astray. You know how unwilling people are to confess, I mean to themselves as well as to the doctor, that they are consumptive. The history of blood-spitting, of a previous attack of consumption, and especially of family tuberculosis has to be dragged out of them. We must laboriously and repeatedly enquire into the past history of our cases and for the occurrence of tuberculosis in different organs. Secondly, we must make a careful investigation of the present symptoms and signs with the deliberate intention to distinguish old and quiescent from recent and active lesions, or to unravel, as it were, the relative extent of the two forms in the case before us.

I will not recount to you what I began with, the appearance of the patient and physical signs met with in quiescent cases. And the clinical characters of active tuberculosis are familiar to you. I only say to you in conclusion: Remember that either or both may be present, and that you must take the trouble to distinguish them.

The benefit to be derived by prescribing ergot in combination with strychnine is particularly noticeable in those cases of amenorrhœa with anæmia that resist the exhibition of iron. The tabloid composed of three grains of extract of ergot and one thirtieth of a grain of sulphate of strychnine, just issued by Messrs. Burroughs, Wellcome, and Co., should prove useful in this respect.

Digitized by Google

A CLINICAL DEMONSTRATION AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

MAY 7th, 1901.

By C. O. HAWTHORNE, M.D., M.R.C.P.,

Physician to the Central London Ophthalmic Hospital; Assistant Physician to the Royal Hospital for Children and Women.

A Case of Double Optic Atrophy with Argyll-Robertson Pupils and Absence of the Knee-jerks.

This man is 40 years of age. According to his own judgment he has only a single symptom, viz. gradual failure of vision, now advanced to a condition of almost complete blindness. But whilst it is true that he only makes complaint of this single, but very serious condition, the case presents three objective signs of organic disease, and it is to these objective signs and their clinical significance that your attention may, I think, be usefully directed. In the first place the patient exhibits double optic atrophy. If you examine him with the ophthalmoscope you find that the optic discs are extremely white or slightly greyish, that the edge of each disc is clearly and sharply defined, and that the retinal blood-vessels are somewhat diminished in size. It is necessary to lay stress on the fact that the edges of the discs are very distinct and clean, because this shows that the optic atrophy is not the result of a previously existent optic neuritis. In optic neuritis there is, of course, inflammatory exudation, which causes swelling and obscures more or less completely the edge of the optic disc. In the course of time this inflammatory exudation undergoes organisation, and the newly-formed fibrous tissue, by its contraction, presses on the fibres of the optic nerve and causes them to atrophy. That, therefore, is *post-neuritic atrophy*, and we recognise it by the fact that the edges of the disc are irregular, or blurred, or concealed, and that usually, also, there are white lines of exudation running along the sides of the blood-vessels. But in our patient the edges of the disc are not irregular or blurred. On the contrary, they are exceptionally sharp and clean. The case, therefore, is not one of post-neuritic optic atrophy. The condition is not secondary to inflammation, but is one in which atrophy or decay of the nerve-fibres occurs as a primary fact, and we therefore distinguish it by the term *primary optic atrophy*. That is the first objective sign of organic disease which our patient presents. It is well, I think, to remark here that the diagnosis of optic nerve atrophy should never be made on the appearance of the disc alone. The tint of the healthy optic disc varies in different individuals within fairly wide limits, so that mere pallor of the disc is not a secure basis for a diagnosis of optic nerve atrophy. There must be, in

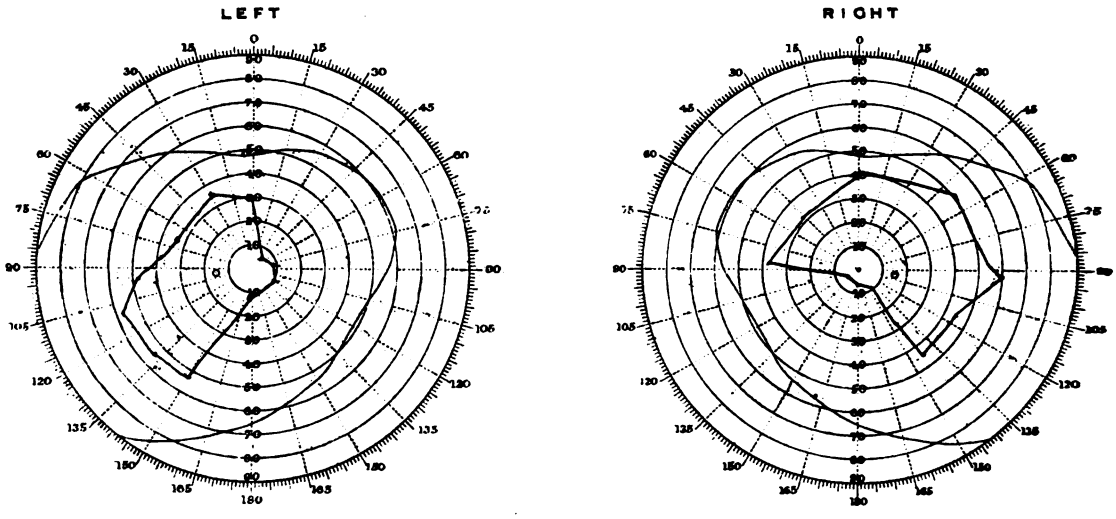


Fig. 1.—Contraction of the visual fields in optic atrophy.

Digitized by Google

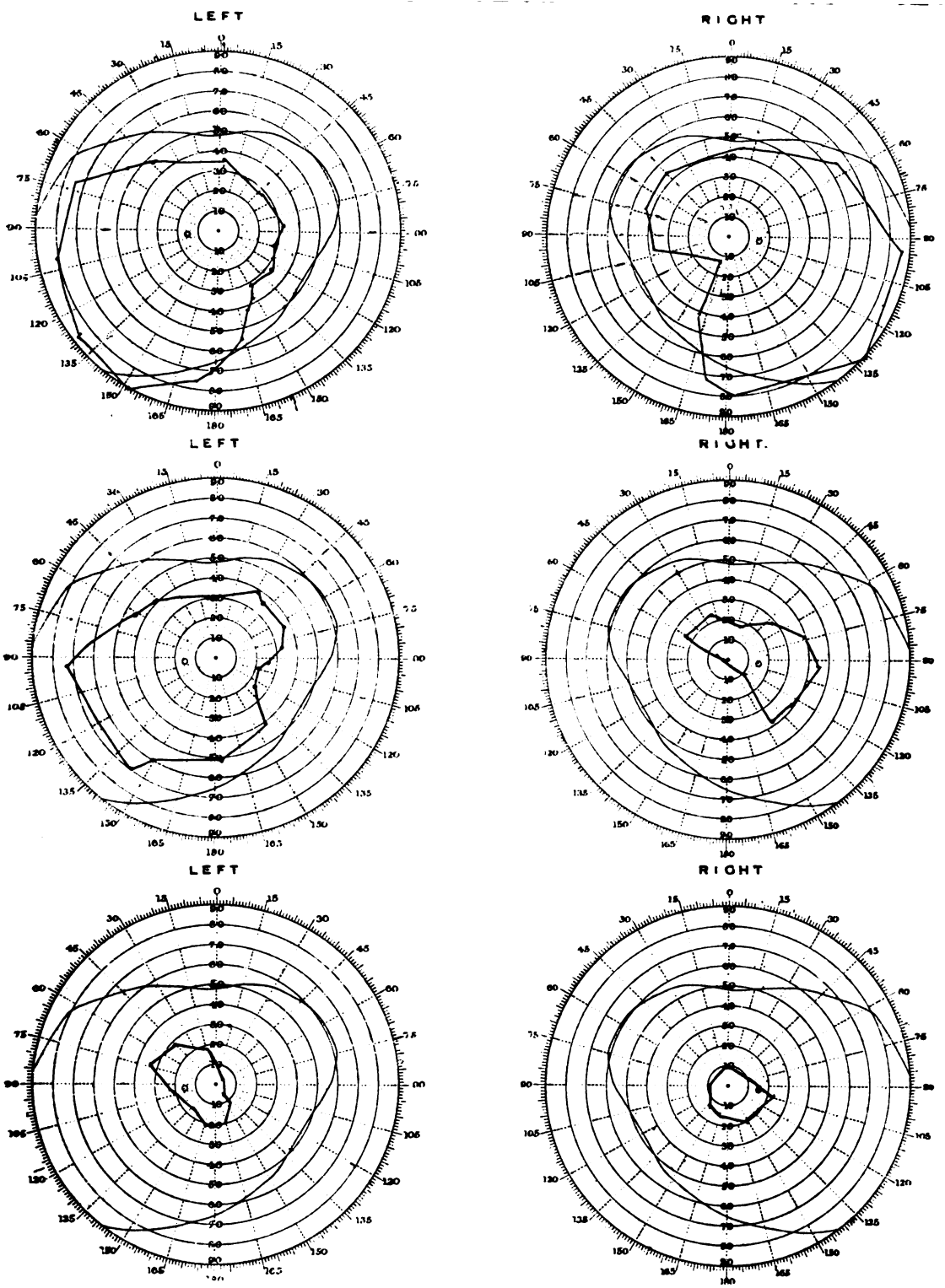


FIG. 2.—Perimeter tracings showing gradual contraction of the visual fields in a case of primary optic atrophy. The first tracings were taken on April 30th, 1898, and the vision was then—Right $\frac{6}{9}$, Left $\frac{6}{6}$ part. On August 13th, the date of the second tracings, the vision was—Right $\frac{6}{6}$, Left $\frac{6}{6}$. Even on October 9th, when, as the third pair of tracings show, the contraction of the visual fields was extreme, central vision as tested by the ordinary types gave—Right $\frac{6}{6}$, Left $\frac{6}{6}$ part.

addition, failure of vision. And here is a very important consideration, namely, that the failure of vision may reveal itself in at least two different ways. It generally, as in the present instance, reveals itself as a defect in the visual power, of the existence of which the patient is only too vividly conscious. But it is quite possible for a measure of optic atrophy to exist and yet the patient have in the ordinary sense of the term fairly good eyesight. That is to say, that at the outset of optic atrophy the patient's capacity for direct or central vision may be nearly or quite up to the normal level. Tested by the standard types it may be concluded that he has quite satisfactory vision. But if you proceed to trace the visual field by means of the perimeter you find that, though central vision is almost or altogether unaffected, peripheral vision has begun to decline, as is shown by the contraction of the limits of the field. Though our patient is now practically blind, he had, when he first came under observation, a fair measure of direct or central vision. But there could be no doubt about the diagnosis of optic atrophy, because, in addition to the appearance of the discs, the perimeter tracings, as these charts show you (Fig. 1), detected considerable contraction of the visual field. Here are the visual fields of another patient (Fig. 2), taken at different stages of his disease. At the date of the first observation the man had nearly normal central vision. Yet even then, as the first tracings show, his visual fields were beginning to undergo concentric contraction, and this, together with the condition of the discs, was quite sufficient to justify a diagnosis of optic nerve atrophy. Subsequently, as you see from the later tracings, the visual fields became smaller and smaller, central vision also began to fail, and in the end the patient became nearly blind. The points I wish now to emphasise are, first, that for the diagnosis of optic atrophy there must be not only pallor of the disc, but also failure of vision; and, secondly, that to detect that failure, at least in early cases, it may be necessary, in addition to the estimation of the power of direct vision by means of the usual types, to test the extent of the visual field by means of the perimeter. In our present patient we can have no doubt there is optic nerve atrophy, because not only are the discs extremely white, but both central and peripheral vision are practically abolished; and for the reasons I have already stated, we know the condition to be one of primary, not post-neuritic, optic atrophy.

The second objective evidence of disease which we find in this patient is detected on examination of the pupils. I look at his pupils and see that both are small, and that the left one is rather larger than the right. Now I apply the light test, taking each eye separately, and I find that the pupils fail to respond. I cannot test their movement on convergence in the usual way, because he cannot see my finger or any other object though I hold it close to him. But I tell him to "look" at the point of his nose, and you observe that when he does so, the pupils, already small, become still smaller. There is therefore myosis or contraction of the pupils, loss of the light reflex, but retention of the power of contraction on convergence, that is, the patient exhibits the "Argyll-Robertson pupil." Does that mean organic disease? Possibly some discussion may be excited by that question, but I do not think that any one will dispute the proposition that, if the Argyll-Robertson pupil does not definitely and invariably mean organic disease, it very frequently has this significance, and that in a case like the present, where we have an undoubted organic lesion in the shape of primary optic atrophy, this condition of the pupils means central nervous disease. Further than that, it affords a suggestion as to the situation of a central lesion. As every one knows, the pupil light response is a reflex phenomenon which depends upon the integrity of a reflex arc. The afferent limb of that arc consists of the optic nerve and tract, terminating in a centre in the corpora quadrigemina; the efferent limb is formed by the nucleus and fibres of the third cranial nerve. The arc, however, as you see from this diagram, is not yet complete. It needs a connecting link between the two centres, and this is provided by the nervous strands known as Meynert's fibres. Interrupt the arc at any point and there must be loss of the light reflex. Now with myosis and the Argyll-Robertson pupil the interruption cannot be in the afferent limb, because numbers of patients who have this condition of the pupils enjoy perfectly good sight. This must mean that the whole course of the optic tract, from the retina through the optic nerve, corpora quadrigemina, and optic radiations to the true visual centre in the occipital lobe, is intact. Neither can there be

Digitized by Google

an interruption in the efferent limb, because were the nucleus or fibres of the third nerve destroyed, the pupils would be dilated, and further, the patient would not display contraction of the pupils in the act of convergence. But there must be an interruption of the reflex arc at some point. And if you place the interruption in Meynert's fibres you can understand how the light reflex is destroyed, and yet the patient has both an unbroken tract through which sensory impressions may reach his cerebrum, and an intact motor mechanism by which the pupils may be contracted when the eyeballs are converged in looking at near objects.

It is interesting to notice that, as our patient demonstrates to us, we may have a contracted pupil associated with optic nerve atrophy. At first sight that seems an extraordinary combination, because in optic atrophy the path along which light impressions travel is destroyed, and it is these light impressions acting reflexly on the centre which excite contraction of the pupil. Therefore, one would expect that in optic atrophy the pupil would be dilated. Frequently this is the case; but, as we see in this instance, it is not always so. Here we have optic nerve atrophy together with a contracted pupil. How does this come about? Remember that we have not merely a contracted pupil. We have also an Argyll-Robertson pupil. And an Argyll-Robertson pupil suggests a lesion involving Meynert's fibres, that is a lesion in the immediate neighbourhood of the third nerve nucleus. That nucleus comprises a group of centres. One of these centres discharges impulses which produce contraction of the pupil. Another leads to inhibition of the pupil-contracting mechanism, and so, when active, produces dilatation of the pupil. Now it is not difficult to imagine that a lesion in the neighbourhood of Meynert's fibres may modify the activity of one of these centres. Either by irritation of the pupil-contracting centre, or by destruction of the pupil-inhibiting centre, it may readily lead, as in the present case, to a small and contracted pupil. Thus we may conclude that here we have to deal not only with an atrophy of the optic nerve fibres but also with a central lesion, and that the latter explains both the Argyll-Robertson phenomenon and the contraction of the pupil.

I wish now to demonstrate the third objective sign of organic disease which our patient exhibits. It is the absence of the knee-jerk. Before we come to the conclusion that the knee-jerk is absent in this or any other case we must exhaust all possible methods of examination, and it is well, before accepting the negative as proved, to repeat the tests on more than one occasion. In a superficial examination it is very easy in some patients to fail to arouse the knee-jerk, even though it is potentially present. I now test this patient in the usual way: I repeat the test, applying the stroke to the bare skin —which is an absolute necessity before concluding that the jerk is absent; I try the method of "reinforcement"; I adopt the plan associated with the name of Dr. Buzzard, in which we appreciate the response in the shape of a contraction of the vastus internus felt by the hand placed on the thigh; and lastly, I endeavour to excite the jerk with the patient sitting on a table with his legs "dangling" over the side so that the hamstring muscles are thoroughly relaxed. In each and every instance the result is negative, and as the same result has followed on previous occasions, we are justified in saying that the knee-jerks are entirely absent. Here then are three definitely abnormal conditions, viz.: optic atrophy, myosis with Argyll-Robertson pupils, and absence of the knee-jerks. What is the significance of such a combination?

There can be no possible doubt that these three facts mean organic disease, and further, we must, I think, allow that they mean organic disease belonging to the group of cases which we know clinically as tabes dorsalis or locomotor ataxia. It is quite true that this man exhibits no evidences of ataxia. As you see, making some little allowance for his blindness, he walks across the room without unsteadiness. Even when made to walk backwards there is little or nothing in his gait to attract attention. There is no swaying when he stands with his eyes closed and his feet close together. As he unbuttons and buttons his coat and vest you observe that he enjoys successful co-ordination of the muscular movements of his upper limbs. There is none of the hesitation and fumbling such as you see in persons with ataxia of the upper limbs. His muscular sense, as tested by calling upon him to state the position of his limbs when these are placed in various

Digitized by Google

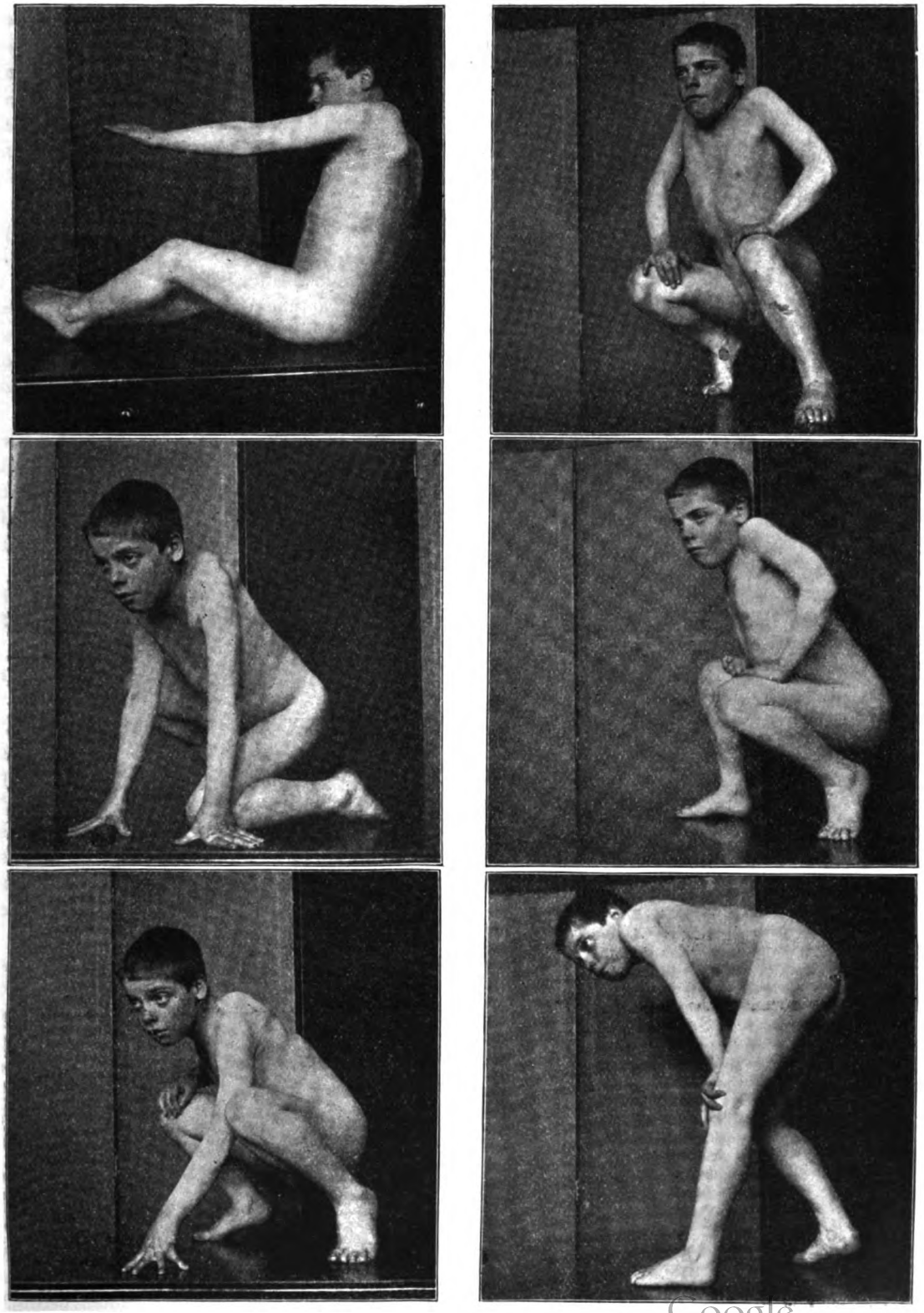
situations, is perfect. There are, indeed, no spinal symptoms whatever other than the loss of the knee-jerks. He has no lightning pains, no disordered sensation, no girdle sensation, the functions of the bladder are perfectly performed, the bowels act daily, and the sexual power is unimpaired. Indeed, the only symptom he will allow, other than his blindness, is a certain sense of pressure in the region of the perinæum. That is of some value in the diagnosis. Patients afflicted with the ordinary form of locomotor ataxia, as many of you must have observed, occasionally suffer from obstinate constipation, sometimes associated with a sense of pressure or discomfort, as from the presence of a foreign body in the rectum. So I claim this symptom of fulness or pressure in the perinæal region, which our patient admits, as favouring the diagnosis I have suggested to you. Further, we have the certain facts of the double optic atrophy and the Argyll-Robertson pupil. Now we know that the Argyll-Robertson pupil with myosis frequently, and optic-nerve atrophy occasionally, are events in the course of locomotor ataxia. The first of these is often an early event in the disease. Optic atrophy is more usual, perhaps, at a later stage. But it is quite possible for optic atrophy to occur at an early stage, and further, it is a matter of relatively frequent experience that when such is the case, the spinal symptoms are slight in degree, and are slow to develop. Indeed, the spinal symptoms in such cases may be, and frequently are, indefinitely delayed. I therefore say that we must attach to this case the diagnosis of double optic atrophy, with myosis and Argyll-Robertson pupils, and absence of the knee-jerks, probably belonging to the tabes dorsalis group. From what I have already said, whilst the prognosis as far as his eyesight is concerned is as bad as it well can be, the outlook in reference to spinal symptoms is relatively good. The very extremity of his ocular condition justifies us in anticipating that the progress of the disease in the spinal cord will be slow, and possibly or probably will not develop beyond its present stage. There is no history or evidence of syphilis; but the prevailing current of opinion would regard the condition as the result of specific disease. Mercury and potassium iodide given in fairly heroic doses at the outset of the optic atrophy have failed to produce any benefit. It would be unwise, I think, to push them further. He is now taking a course of arsenic and strychnine, and this is alternated with full doses of cod liver oil. In this way, I hope, we are doing something to preserve the unaffected portions of his nervous apparatus, and to assist what appears to be the natural tendency to postpone or avert the development of spinal symptoms.

Perhaps it is worth while remarking, in connection with the diagnosis of the case, that a combination of the Argyll-Robertson pupil and loss of the knee-jerks is not infrequent in an early stage of some cases of general paralysis of the insane. Even if such a question could have been raised when the patient was first seen, some six months ago, it certainly is not possible now. He has none of the mental or motor phenomena of general paralysis. His memory and general mental capacity remain unaffected. His tongue when protruded exhibits some slight rhythmical tremor, but in showing his teeth there is none of the uneasy facial twitching which general paralytics so often exhibit. The mere lapse of time without the development of any of these symptoms, apart from other considerations, makes it certain that the case is not one of general paralysis.

A Case of Pseudo-hypertrophic Paralysis.

Here is a little boy æt. 5 years. He was brought to me a year ago with the statement that he had never walked, and that he would not make any attempt to walk. He appeared then a fairly healthy-looking boy, but on endeavouring to get him to stand, his legs seemed to collapse under him, and he objected to the experiment. He had no deformity of the spine or lower limbs, the knee-jerks and superficial reflexes were present, and the only question which was raised at the first observation was whether the muscles of the calves and buttocks were not unduly large and firm, more especially as these had not enjoyed the usual discipline of walking exercise. Let us look at him now. He is evidently a somewhat pale and timid boy, but at first sight he presents nothing very remarkable. I ask his mother to strip him, and now, as he stands up supporting himself against the chair, you notice two peculiarities. First, he keeps his feet widely apart, so as to get a wide basis of support. Secondly, there is very decided lordosis. When he sits down the lordosis

Digitized by Google



Digitized by Google

FIG. 3.—Pseudo-hypertrophic paralysis. Series of photographs (to be read from top to bottom) showing the successive stages in the movement of rising from the ground into the erect position. (I am indebted for these

disappears, and indeed his lumbar spine becomes convex posteriorly, as if from weakness of the erector spinæ muscles. He now walks for a number of yards but with a very peculiar gait. As you see he keeps his feet widely apart; his body sways from side to side; and as he advances his limb he tilts up the side of his pelvis so that he may swing the foot clear of the ground. I next place him flat on his back and ask you to observe the way in which he rises. First he rolls himself over on to one side, then, using his knees as a fixed point, he manages to get on to his hands and knees. Now, depressing his head, his hands travel backwards towards his knees. Here, in consequence of the weakness of the muscles of the back, he is unable to straighten his spine, and to do this he gets a hand on each knee, and "climbing" with his hands upwards along the thighs manages, as it were, to push himself into the erect position (Fig. 3). The attitude, the gait, and this last series of manœuvres which he has just displayed to us, are all noteworthy in the highest degree. Let us now examine the muscles. As you see he has very creditable calves and well-developed buttocks. But when you handle these regions you feel at once that they have not the elastic feeling of healthy muscle, but that they yield a hard, inelastic, wooden sensation. And the calf muscles are not strong, as their bulk would at first sight suggest; for the boy, when I tempt him with a penny held above his head, cannot rise on tip-toe to take it. This enlargement of the buttocks and calves is all the more noteworthy when you examine the muscular condition of the upper limbs, which have enjoyed the ordinary activities of childhood. If you examine the muscular masses about the shoulders you will have no doubt that these are wasted or imperfectly developed. And as I lift the boy up by his arm-pits his shoulders go up almost to his ears, as if there were no proper power in such muscles as the pectoralis major and the latissimus dorsi which depress the humerus. The trapezius and the serratus magnus seem fairly normal, but the muscular clothing of the scapulæ is certainly defective. So far as I know the infraspinatus muscle has never appeared large, but it sometimes is affected in just the same way as the calf muscles. The diagnosis is obviously that of pseudo-hypertrophic paralysis, and it rests on the following grounds.

First, there is the age of the child. The disease is a congenital one, but it usually begins to manifest itself rather later than in this case, that is after the child has been walking and running about in the usual fashion for some few years. But occasionally, as in the present instance, pseudo-hypertrophic paralysis develops before the child has ever attempted to walk, and this is apt to lead to a mistake in both diagnosis and prognosis. Every practitioner sees cases in which, whilst the inclination to walk is postponed, the child after a time walks perfectly well. Hence when a healthy child of two or three years is brought with a story that he will not walk, and you find him free from deformity and with well-developed limbs, there is an inclination to prophesy smoothly, and to say that in a short time all will be well. And yet, as here, the future may provide a very painful contradiction. Hence in dealing with children in whom the walking function is postponed it is necessary to think of the possibility of pseudo-hypertrophic paralysis.

The next element in the diagnosis is the sex of the patient. The disease is very much more frequent in boys than girls.

Thirdly, we have the characteristic attitude, gait, and manner of rising from the ground.

In the fourth place there is the hypertrophy, or rather the pseudo-hypertrophy, of some muscles and the atrophy of others. The enlarged muscles, of course, are not really hypertrophied. The muscular elements, indeed, are defective, as we see in the calf muscles in this boy, which he cannot use to raise himself on tip-toe. The enlargement is due to increase of connective tissue with more or less adipose tissue.

And lastly, to complete the diagnosis, there are the electrical reactions, the affected muscles showing a defective response to both galvanic and faradic currents.

These are the grounds on which the diagnosis of pseudo-hypertrophic paralysis rests.

The case presents individual features which are somewhat noteworthy. The early occurrence of the symptoms I have already commented on. Then there are the evidences of improvement. When first seen he could not walk at all; now he crosses at least a dozen yards, and nothing offends him so much as to say he cannot walk. It is sometimes said that in pseudo-hypertrophic

Digitized by Google

paralysis there is never any improvement. That is, I think, a hard saying; it is contradicted by the present case, and by other cases I have seen. Another fact to be recorded is the absence of any family history of the disease. The patient's father, I am told, did not walk until he was three years of age, but this can scarcely have any bearing on the present condition of his son. Whilst the disease is regarded as a family tendency, the transmission is through the female line. But in none of the mother's male relations has there been any such condition as the present. And our patient is her only male child; there are two other children, but they are girls, and are quite well and strong. It is true that the family inheritance is often conspicuous, but isolated cases occur, like the present, not very infrequently. Lastly, I ask you to notice that the knee-jerks are present. They may be absent in pseudo-hypertrophic paralysis, but only when the quadriceps extensor is affected. The disease does not depend on a spinal lesion, but is a muscular dystrophy. Hence, if the muscle on which the knee-jerk immediately depends is not affected, the knee-jerk is retained. That is the case here.

It is impossible to give a hopeful prognosis, and all we can do for treatment is to promote his general nutrition, and to give his muscles as much exercise as possible without making him tired. These children should be kept walking about as long as possible. Ultimately, it is to be feared, the boy will become bed-ridden, and some intercurrent disorder, such as bronchitis, or pneumonia, or phthisis, will lead to a fatal end.

A Case of Coloboma of Each Iris.

This girl is the subject of anæmia and dyspepsia, with which we need not just now concern ourselves. What I wish you to observe is the condition of her pupils. There is, as you see, a gap or deficiency in the lower inner quadrant of each iris. She has a coloboma of each iris. That is a defect of development. It is due to imperfect closure of the choroidal cleft, and the position of this cleft explains how it is that the coloboma is always in the position which we find it to be here.

A Specimen of White Urates.

Here is a specimen of urine containing an abundant white deposit. We so frequently see pus or phosphates as a white deposit, and urates as a fawn-coloured or reddish precipitate, that we are apt to forget that urates may sometimes be white. The deposit here consists of urates, for it dissolves on heating or on the addition of liquor potassæ. The specimen has been obtained in the circumstances in which white urates usually, I think, occur. A young child is brought with a complaint that it screams when making water. On examination, the urethra and neighbouring parts are found to be normal, but the urine is highly acid, and it deposits copious urates, which are often, as in this instance, almost or completely white. The deposit may be amorphous, or it may contain "hedgehog" crystals of urate of sodium. A few full doses of potassium bicarbonate corrects the hyperacidity of the urine, and cures your patient.

Blows on the Head.—Experiments show that the normal brain pressure, as evidenced by the bulging of the uninjured brain of an etherized cat through an opening left after removal of dura and bone, unless pressure is applied to keep it in place, is increased by every factor increasing the general blood-pressure. A cry (raising thoracic pressure), any general muscular movement, stimulation of the sciatic nerve, the administration of ether (increasing the heart's action), all demand a stronger pressure to keep the brain level. Pulsations after hæmorrhage are increased, but the actual brain pressure is less. After heavy blows on the skull, the pressure needed to prevent the brain from bulging is greatly increased. Experiments thus far fail to show that there is any increase of general arterial pressure to account for the greater brain pressure after injury. No observations have, as yet, been made to ascertain if the increase is to any degree dependent on increased cerebral *venous* pressure. The practical application of these experiments lies in the confirmation of the view that the *respiratory centre is the first to be affected after a blow on the head.* Many times when respiration has ceased, artificial respiration for a few minutes has been followed by normal breathing, which has continued for hours.—*New York Med. Journ.*, June 22nd, 1901.

Digitized by Google

ARSENICAL POISONING FROM BEER DRINKING.

By T. N. KELYNACK, M.D., M.R.C.P.

RECENT experiences clearly indicate that arsenical contamination of beer may lead to calamitous results in the drinkers of such a beverage. During the recent "epidemic," in many instances death has ensued, and in a vast number of cases incalculable suffering and serious bodily ill have resulted. There is strong reason to believe that for some time past many of the so-called "malt liquors" have, at least in certain parts of the country, contained arsenic, and doubtless some of the cases we have been accustomed to consider "alcoholic paralysis" have been in part or mainly due to this agent.* Now that the danger arising from the introduction of arsenic into beer and stout and food products has been clearly recognised, it may be hoped that the State will take steps to adequately protect us from the dangers incident to such contamination. But since history is wont to repeat itself, it will in future be incumbent on every medical man not only to be fully cognisant of the danger, but to be quick in recognising evidences of arsenical poisoning.

It will therefore be well, while the facts of the recent outbreak are fresh in the memory, and before the sufferers have altogether passed from under observation, to briefly review the more important clinical manifestations.

The history of the recent outbreak † is now so well known, and many of the points still under discussion being chiefly of pathological and medico-legal interest, it will be best to limit our consideration to purely clinical features. And it will be most convenient for our present purpose to consider the signs and symptoms as they have occurred in connection with what we are accustomed to term the various systems of the body.

The cutaneous lesions are particularly conspicous and very multiform.

Pigmentation (melanosis arsenicale) is one of the most marked and characteristic of the cutaneous manifestations. It varies from a slight dirty tint to the darkness of a mulatto, and often closely resembles the "vagabond's pigmentation." It is generally well marked over normally pigmented areas, but exposed parts, and even the greater portions of the body are often darkened. The genitals, perinæum, and abdomen are frequently most affected. The areolæ of the nipples are darkened and sometimes quite black. The mucous membranes are never pigmented. The distribution of the pigmentation frequently gives a mottled or spotted appearance, such as is never met with in Addison's disease.

Erythematous lesions are common, and most frequently affect the distal portions of the extremities. They may be of a diffuse scarlatiniform or morbilliform character. In many instances the erythema is associated with marked pain, constituting the affection designated erythromelalgia.*

Papular, vesicular, and bullous eruptions occur. The papules are often accompanied by pruritus, and hence tend to become associated with scratch lesions.

Herpes when present is of diagnostic significance. The formation of blebs is exceptional, but large blisters may form in the thickened cuticle about the feet.

Pustular lesions also occur. Boils are met with in a few cases, especially in the axillæ and about the genitals and buttocks.

Desquamation is a common feature. The exfoliation of the skin in some occurs as a mere branny shedding, in others the cuticle separates in large flakes. Arsenic can be readily detected in this separated skin.

Keratosis, or thickening of the cuticle (hyperkeratinization), long known as a characteristic effect of arsenic, is a conspicuous feature in a large number of cases, and principally involves the hands and feet.

Psoriasis-like lesions occur occasionally on the extremities and even on the trunk.

Hyperidrosis, or excessive sweating, may be a marked feature. The cuticle in these cases often has a macerated appearance.

Localised œdema is occasionally met with.

Alterations in the nails and hair also occur. The nails become hard and brittle, and may be

* See 'Medical Magazine,' July, 1901.

† See 'Arsenical Poisoning in Beer Drinkers,' by T. N. Kelynack and W. Kirkby, London, 1901.

* See the 'Clinical Journal,' June 19th, 1901.

Digitized by Google

shed. In many the growth of the nails is rapid, and some show transverse ridging. The hair in many becomes detached in considerable quantities. It is of interest to remember that the presence of arsenic can be readily detected in the hair of these cases.

The most conspicuous manifestations of the poisonous action of the arsenic are met with in connection with the nervous system. Most of these indicate peripheral involvement, but some point to changes in the cord itself, and in some few there are evidences of cerebral disturbance.

Sensory disturbances usually appear earliest and are marked. They are of almost all varieties and vary much in degree.

Cutaneous hyperæsthesia is a marked feature, often intense, and in many cases exceedingly persistent. I have known it continue for upwards of a year after all beer was stopped. Muscular hyperæsthesia is frequent. Hyperalgesia is sometimes present. Anæsthesia is sometimes met with in the more chronic cases. Analgesia occurs, but is exceptional.

Neuralgic attacks of pain not infrequently occur in the course of the affected nerves. The shooting or lancinating character has even suggested locomotor ataxia. Pressure over the nerve trunks sometimes causes intense pain.

Paræsthesiæ are common. Sensations as of "pins and needles," "numbness," and the like, are constantly complained of. Usually they are limited to the hands and feet.

Thermo-anæsthesia can often be detected. Delay in the rate of sensory conduction is noticeable in many. There is often impairment of the muscular sense. Touch perception may be diminished or lost.

Motor derangement is present in nearly all instances. In some it may be comparatively slight, but in severe cases it leads through varying degrees of paresis to complete paralysis.

The muscles of the extremities suffer most. There is lessened power, loss of tone, and marked wasting. Many show distinct incoordination. The reaction of degeneration is obtained in severe cases.

The lower limbs usually suffer most, and are slowest in recovering. The degree of paralysis varies greatly. The extensors are most affected. The small muscles of the hand are also often much involved. Dropped hands and feet are common in bad cases.

Sometimes the abdominal and back muscles are involved, and the diaphragm may be paralysed.

Tremor is sometimes present, but is not a conspicuous feature. Convulsions are exceptional. Cramp is often most distressing. This is often situated in the calf as in ordinary alcoholics, but in these cases it is frequently localised in the region of the toes, and particularly about the extensor tendons of the great toe. Contractures call for careful management in severe and long-continuing cases.

Alteration of reflexes varies much with the severity of the case and the extent of involvement.

The superficial reflexes are often exaggerated, and may long continue so. When lost the plantar reflex is often the first to return. In bad cases all forms of superficial reflex may be lost.

Of the deep reflexes the knee-jerk is generally absent in severe cases. In mild, and in the early stages of acute cases, it may be exaggerated.

The visceral reflexes may be deranged. Difficulty in micturition is sometimes experienced, but would seem to be generally dependent on local conditions.

Trophic affections. Many of the cutaneous lesions, such as herpes and the pemphigoid and urticaria-like eruptions, are to be looked upon as of tropho-neurotic origin. Glossy skin is sometimes present. Trophic changes also occur in connection with the nails, and sometimes with the hair. A tendency to develop bedsores is noticed in many cases.

Psychical manifestations are occasionally present. Hallucinations, chiefly visual, are sometimes experienced. The more noteworthy mental derangements, however, are such as are frequent in alcoholic subjects.

Respiratory affections seem chiefly to consist in a catarrhal condition. There is serous discharge from the nose, and congestion or actual catarrhal inflammation of the mucous membrane. The larynx is frequently involved, and the voice rendered husky. Phonation may be difficult or almost impossible. Irritable cough is common, and tracheal and bronchial catarrh frequent. The lungs do not appear to suffer primarily to any extent.

It is well to remember, however, that paresis of

Digitized by Google

the intercostal muscles, and even complete paralysis of the diaphragm may occur. Occasionally vague thoracic pains are complained of.

In regard to circulatory derangements, the cardiac enlargement so frequent in beer-drinkers is, of course, present in many of the cases. In many there is distinct evidence of muscle failure. The cardiac asthenia is sometimes a conspicuous feature. The pulse in bad cases is quick, of low tension, and readily influenced by alterations in posture. Anæmia occurs in many cases. Slight leucocytosis has been noticed in some instances.

Concerning digestive disorders, evidences of irritation of the gastro-intestinal tract are present in a number of cases. Where the amount of arsenic in the beer is considerable vomiting and diarrhœa occur, associated often with some abdominal pain. In a large number of cases, however, no gastric derangement is met with.

In some few instances increase of appetite and a sense of well-being have preceded the onset of symptoms.

But nausea and anorexia are often present. The tongue is frequently furred, but rarely silvery. The mucous membrane of the mouth seems never to be pigmented. In many constipation is most persistent.

Occasionally slight discomfort in the urinary tract is complained of. Micturition may be accompanied by smarting. Cystitis is occasionally met with. Retention of urine may call for catheterisation. The urine is often high-coloured, and may contain albumen. Arsenic may be readily detected in the urine of many of these cases. In some cases there has been some irritation about the genitals.

It is thus seen that the assemblage of symptoms, although clearly indicative of arsenical poisoning, has in many respects presented features differing somewhat from the hitherto customary cases of arsenical poisoning.

Bacteriology of the Urine (Dr. Lindley Scott).—Pathogenic urinary bacteria are clinically divided into (1) *those which have the property of decomposing urea*, and give rise to cystitis, pyelitis, and pyelo-nephritis, with an *alkaline* (ammoniacal) urine depositing pus, epithelial cells, and *debris*—these include various staphylococci, streptococci, diplococcus urea liquefaciens, proteus Hauser, etc.; and (2) *those which do not possess the property of decomposing urea*, and cause either simple bacteruria, or cystitis, pyelitis, and pyo-nephritis, with a variable quantity of pus, epithelia, and *debris* in an *acid* urine—these include the tubercle bacillus, typhoid bacillus, gonococcus, and the *Bacillus coli communis*. These organisms find their way into the bladder from the urethra, usually by the passage of instruments, or by creeping along the short and wide urethra of the female; more rarely they are carried to the kidneys by the blood-stream, and, if circumstances are favourable for their growth, they set up septic inflammations of the kidneys or bladder. Urinary infection may therefore ascend from the bladder to the kidney, or descend from the kidney to the bladder; so that cystitis, pyelitis, and pyo-nephritis may reasonably be considered various phases of one inflammation. The *Bacillus coli communis* is regarded by Melchior as the most common cause of cystitis and pyelitis, the urine in such cases being acid in reaction, containing more or less pus, and having a peculiar fœtid odour. Its point of entrance is usually the urethra, especially in females; but under certain conditions, such as diarrhœa, constipation, etc., it seems to be absorbed from its natural habitat, the intestine, and being carried to the kidneys by the circulation, gives rise to an apparently spontaneous bacteruria, or to septic inflammation of the urinary system. Various bacteria may be simultaneously present, and thus it is not uncommon to have an alkaline urine in a naturally acid bacteruria, owing to the co-existence of urea-decomposing and non-urea-decomposing organisms. Recent research work seems to make it very doubtful if there exists a special urea-decomposing organism, such as the micrococcus ureæ.

The above remarks are taken from a very valuable book by Dr. Lindley Scott, published by Messrs. Churchill of 7, Great Marlborough Street, and entitled 'The Clinical Examination of Urine, with an Atlas of Urinary Deposits, including forty-one Original Plates mostly Coloured.' The author in the preface says that his endeavour has been to prepare a ready reference handbook to the clinical examination of the urine, and he has succeeded admirably in his effort. A most refreshing characteristic about this work is the fact that the plates really show what the text says will be found there.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 457. WEDNESDAY, JULY 31, 1901. Vol. XVIII. No. 15.

CONTENTS.

PAGE

*A Clinical Lecture on "Some Neuroses." By Prof. T. CLIFFORD ALLBUTT, M.D., F.R.C.P., F.R.S. 225

*A Clinical Lecture on Chorea or Cerebral Rheumatism. By Sir DYCE DUCKWORTH, M.D., LL.D., F.R.C.P. ... 236

*Clinical Remarks upon the Treatment of Thrombosed Varicose Veins of the Leg. By C. MANSELL MOULLIN, M.D.Oxon., F.R.C.S. 239

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE ON "SOME NEUROSES."

Delivered at the Medical Graduates' College and Polyclinic, May 15th, 1901.

By PROFESSOR T. CLIFFORD ALLBUTT, M.D., F.R.C.P., F.R.S.

GENTLEMEN,—A few months ago I went to hear a lecture upon the planet Mars delivered by my friend and colleague, Sir Robert Ball. He was speaking in reference to the so-called canals upon the face of the planet, and he began in a very graphic way, with chalk and blackboard, to describe the ways of flowing water. Very carefully and artistically he showed how thus and thus water behaves itself by virtue of its own properties and by the properties of the surfaces over which it travels. And then, turning round gravely to us, he said, "In the planet Mars there is absolutely nothing of the kind I have been describing to you." Now after some such fashion as this I propose to lecture to you to-day. When I was asked to address you on the subject of "Neuroses," a subject which, in a more or less subordinate way, is supposed to be mine, I answered that I would do so if the title were put in inverted commas, so that the name at any rate should not be regarded as my own, and if, again, it were understood that I should handle the subject in a more or less destructive way.

I may use the word "neuroses" then in the common loose sense in which it is usually employed to signify those nervous disorders which are disorders rather than diseases; temporary and functional disturbances, dynamic states of the nervous system, rather than local static changes. I do not intend to include or to consider malingering or any form of imposture—whether discovered or undiscovered,—nor shall I consider anæmia or other secondary and associated disorders.

It is rather in respect of diagnosis of "neuroses," and of a somewhat destructive diagnosis, that I venture to speak to-day. About treatment I shall

Digitized by Google

not be able to say much in the time at my disposal, unless it be inferentially. The particular kinds of neurosis about which I shall speak to-day are the neuralgias, neurasthenia, hysteria, hypochondriasis, the milder melancholias, and so forth; but you are aware that in the time at our disposal this afternoon I can touch but very cursorily upon any one of these.

I will begin by pointing out that it is important for us not to mistake these disorders the one for the other, or to fail to discriminate between them; that, for instance, we should not confound hysteria with melancholia or neurasthenia, that we should not mistake either of them for hypochondriasis, and so forth. If we do fail to discriminate between these particular disorders we may not perhaps do more harm than to discredit ourselves somewhat, at any rate in our own opinion. But it is a very serious thing for the patient, his friends, and ourselves if we fail to discriminate between one of these dynamic conditions and some condition of static disease, one to which a more serious interpretation should have been given, or one which would have led us directly to some measures of prompt relief. Using the words in their current sense, if it be important that we should not confound the functional neuroses, it is far more important that we should not fail to distinguish between functional disease and organic. Therefore I desire, in that casual way which only is possible in an hour's discourse, to make some unmethodical remarks on these discriminations between functional nervous disorders and between these and organic diseases, so far as may be.

I would first call your attention to the difficulty in discriminating hysteria from neurasthenia, names which I must not hesitate to use to-day, simply for the sake of brevity. I am not here to defend the use of either of them, but they represent concepts which I have tried to define in the article on Neurasthenia in my 'System of Medicine.' In many respects such symptom groups must be indefinite, especially on their circumferences, where they melt insensibly into each other; still, with obvious limitations, I think we may take these names as fairly well understood.

To contrast neurasthenia with hysteria is a very necessary consideration. Hysteria is not confined to the female sex, nor to adults. In children you may find hysteria abundantly. I daresay some experienced hearers in this room have seen even epidemics of hysteria; for instance, in school children. Nor is the affection confined to the female sex. But you may say, what does the distinction itself matter? Well, it matters partly in order that we may retain our self-respect as accurate observers. But it matters also for this further reason, that while neurasthenia on the one hand, by the method of treatment known under the names of Weir-Mitchell, and of Playfair, is usually an eminently curable malady, hysteria, on the other hand, is eminently incurable. Hysteria is not thus readily dealt with; a hysterical patient might be put through such a course as I have described without deriving any benefit, or such benefit only as may be incidental to better and more methodical government for a time. What is prominent in hysterical persons generally—by no means exclusively—is an excess of inhibition by higher centres over lower. For instance, a patient comes into your room in the morning, and you tell him to make water for you; you watch him, and you may perceive that he is not able then to perform the act. So you say to him, "I see you cannot do it in my presence; I will leave the room," and after you have left the room he passes his urine at once. Now this inhibition is of the essence of hysteria. It is not true to say that the man could have made water in your presence "if he liked;" he could not. These interferences are not only prominent features of hysteria, but exceedingly incurable factors of it. In neurasthenia, on the other hand, such inhibitions are not frequent. Here we have rather to deal with a defect of control, a letting go of lower centres by the higher, which latter, by original lack of capacity, by excessive stress, or by insufficient nourishment, are unequal to the offices of command, and to them we endeavour to restore the blood and the nervous tension by such means as those pursued in the Weir-Mitchell system. This restoration is simpler and more easy of attainment than to divert irregular usurpations. But unfortunately for us and himself, a person may suffer from some combination of these two maladies. Such a person may come to you with a history of mental stress or bodily stress, and may, especially if the patient be a woman, combine hysterical symptoms with neurasthenic sym-

Digitized by Google

ptoms, the two co-existing. During the last six weeks I have had two ladies under my care in whom a decisive diagnosis was almost impossible, because the two conditions co-existed in uncertain proportions. All I could say in each case was that we would try to clear up some part of the patient's case at any rate; that we would do our best to clear up the neurasthenia, but in respect of the hysterical element I could make no confident promises. And as I had led the friends to anticipate, we found the cure of the hysterical part beyond our powers. The neurasthenic element in both was quickly relieved, or indeed, cured, by the Weir-Mitchell treatment; whereas the very large hysterical element, consisting in one case of hysterical fits, which are not so common in England as they are in France, was practically untouched by this method.

But far more serious difficulties arise when we have, as we may have, combinations of neurasthenia, or of hysteria, or of both, with organic disease. Then we come upon a group of most embarrassing cases. It is far more alarming to a patient and to his friends to be told of organic disease, or of a combination of neurasthenia or hysteria with organic disease, than to be told of any combination of functional disorders. Personally, indeed, there are few diseases in which I feel more powerless than in hysteria. I would much rather have a case of phthisis, in an early stage, to cure than hysteria. But the public does not think so; the public regards hysteria as a piece of factitious trumpery which a woman can be scolded or drilled out of. I wish, indeed, it were so. If, however, we report hysteria or neurasthenia only when some organic disease is present also, or indeed when the case is wholly one of organic disease, we lay ourselves open to severe blame. Yet hysterical symptoms may so overlay organic disease that only by considerable experience are we enabled to detach or distinguish the two. In many, very many, such cases there is a core of organic disease; such as cardiac degeneration, dilatation of the stomach, chronic degeneration of nerve centres, a dislocation of some viscus, an abnormal adhesion of one part to another, and so forth; of whatever nature and position it may be, there is often, I say, some inward static disease which happens to be concealed in the individual by an embroidery of neurotic manifestations. This atmosphere of functional disorder we have to penetrate if we can. It is in the hope of saying something to help you to disentangle such complex morbid conditions that I venture to occupy your time to-day. I repeat that patients may come to you with one or other of these multiform combinations, and often, too, with a history of stress or of hereditary neurotic conditions which, if the case contain organic disease, will make it the more difficult for you to disentangle the various strands of the coil. Such neurotic perturbations may mystify you in any rank of life, in persons of any calling, and, I believe, in almost any race of mankind.

Let us now pursue these matters a little more into detail. One of the most useful introductions to our knowledge of them was the occurrence and due appreciation of many of the so-called "railway cases." It so happened that at Leeds six or seven large railway companies converged upon the town; so that before the introduction of the block and other improvements in signalling, the number of railway cases in this district was very great, and accordingly at an early date our experience became very large. Yet it was by very slow degrees, and by the ungrateful help of the lawyer, that we were enabled to disentangle the obscure and yet very serious consequences of certain railway shocks, which, so far as we could discover, had inflicted no obvious or "organic" injury upon the sufferer. I may not go fully into this story now; I only refer to it for illustrative purposes. Now medical men concerned in these cases were put into the witness box, and cross-examined by counsel. These counsel became very clever in the subject; they read up their text-books and insisted upon looking behind the technical words which we were apt to use somewhat too confidently. Many of us had not been driven to look behind abstract words in the way these inquisitive lawyers insisted upon our doing. When we tried to ride off on abstract notions the lawyer stopped us to verify and test many steps in the inference, and thus compelled us to undergo searching *vivâ voce* examinations, and to marshal our facts and opinions with a new and very rigorous discipline. In this way it was that the recognition of "railway spine," as it was called,—really a form of neurasthenia,—was established; and was compared with various other forms, such as visceral, cerebral neurasthenia, and so on, until most of these cases were disentangled from chaos.

Digitized by Google

I will now turn to a very terrible, and often, in its early stages, latent disease; namely, General paralysis of the insane. General paralysis is so terrible a disease, that for us to mistake it, but for a day, for anything else must be an unfortunate error. But bear in mind you may be asked to see the patient once only, and you may have no opportunity of revising your diagnosis. Now take a case of this kind—and in addressing you to-day I shall always speak within my own experience. You are sent for, let us say, to see a commercial man engaged in a vexatious business in a large town. He is as yet perfectly sane, but he is good for nothing. He says he cannot walk to his office and back, though it may not be more than half a mile or a mile away. He is fretful, sleepless, and depressed in spirits. Some of his friends say he is "faddy;" the more sympathetic of them say he is overworked, and that his liver is out of order. You, too, may think that he has been overworked, and moreover that he has led a rather unphysiological life,—working late at nights in an unventilated office, not taking proper meals, or taking too many of them with too little exercise. He may have primed himself with alcohol, but whether to great excess or not we have usually not sufficient evidence to decide. The man is run down, but you cannot discover any definite signs of disease. Perhaps, however, he says that once or twice on getting up from the table rather suddenly he has turned giddy, and has had to hold on to the furniture; otherwise it is the ordinary story of fretfulness and irritability; he cannot sleep at night, he is tired by short walks, and after he has added up a few columns of figures, or written three or four letters, he can do no more, and has to go home; in other words a common story of neurasthenia. Now I am bound to say that in the case which I now have in my mind, although I saw the patient three or four times at this stage, and saw him carefully at home, it did not occur to me at first that the case was anything more than an ordinary instance of neurasthenia. The iris was normal, and so was the optic nerve. Thus even eighteen months or two years went by, during which I saw little of him; and then in a moment the diagnosis that the case was one of General paralysis of the insane was made for me by telephone; for this gentleman's son, telephoning to me, said, "My father, whom you may remember, has gone off abroad, without saying a word to anybody, which is quite different from his ordinary habits; and he is drawing money from the firm at the rate of twenty pounds a week more than usual. He has been reminded by his partners that he is spending more than he can well afford, but he has written back to 'pooh pooh' their scruples." Now such a message as this warns us plainly what we may be in for. As promptly I advised the son to scamper off to find his father, and by some stratagem to bring him home as quickly as possible. When the patient arrived at home, we realised with some self-dissatisfaction that those transient vertiginous attacks were the little fits of General paralysis. Yet there was still no affection of speech, no affection of the facial muscles, nothing characteristic to establish this fatal diagnosis. We had said to ourselves, and to the patient's friends, that neurasthenic people are apt to have vertigo; yet their vertigo is a vaguer affection, more persistent and more subjective. If a man has a sudden vertigo, which passes off again as quickly, it must not be assumed to be a neurasthenic vertigo which the man talks about, but the effects of which are not seen. Of this patient it was said by the family that once or twice, had he not caught hold of something, he would have fallen, though there was never an instant's loss of consciousness; and this was the only key, I believe, to the diagnosis of the case I have indicated. Those who have seen much of General paralysis know it may go on for a much longer time than the three years of the text-books. It is now six years since I first visited this patient; he is surviving now in the third stage of general paralysis; in a pitiable state, it is true, but still alive.

Perhaps in this case one or two things might have put me better on my guard. The man lamented his "neurotic" condition, it is true, and was melancholic about it; wrongheadedness and extravagance appeared much later. A neurasthenic patient is always willing and wishful to correct his errors; he will write letters, and he will leave out words in them, but he will remember his failing and complain that he is obliged to read his letters over again laboriously in order to correct them. I suspect the general paralytic, even in the earliest stages, does not do this. Again, there is something more docile about the neurasthenic patient; not only is the general paralytic apt not to correct his

Digitized by Google

shortcomings; even in the earliest stages he lacks self-criticism, as in the later he lacks repentance and remorse. Yet after all, perhaps, some of these cases in a very early stage are undiagnosable. I have seen many others which were far from easy to read, either by myself or by others yet more expert in mental diseases.

Let me now say a few words on cerebral tumours. It may seem strange to you that cerebral tumour may be mistaken for a "functional neurosis," or *vice versâ*, but such doubtful cases are not rare. About three years ago I saw a lady's maid of some forty-five years of age, who had become "hysterical"; she declared she could not do this and that and the other simple task; she had become fanciful and "queer," but there was little else to be made out about her. Her friends proposed that common and futile diagnosis of "change of life." When I saw her with the family attendant I had this further advantage in diagnosis, that while under my eye and submitting to my examination, she leaned back in her chair and went off to sleep, till roused by louder questioning. I found nothing then wrong with the optic discs, and there was no headache or vomiting; but I thought it better to take her into the Addenbrooke's Hospital. Within three or four days optic neuritis began, and within two or three weeks she died of a tumour in the left anterior lobe, which proved to be of the size of a hen's egg. For some time, for many years perhaps, this tumour had been growing silently before some slight symptoms appeared which, very naturally, had been taken to signify hysterical neurasthenia; indeed, till near her end there was no symptom, such as headache or optic neuritis, to suggest a graver opinion. The peculiar occasional somnolence had not come on till the last stage of her disease set in; and within a week or two after the appearance of optic neuritis she died. I may refer to another case, not under my own care, one in which the patient, a person of fifty or fifty-five years of age, had complained for some time of severe recurrent headache, and with that headache sickness. Two medical men had given a definite opinion that the case was one of migraine, due to over-work. Now it turned out to be one of cerebral tumour, and very great blame was thrown upon the medical men because the true nature of the case had not been declared sooner. But the only criticism I myself would venture to make upon this diagnosis is that migraine does not begin in middle life; nay, I would go further than this, and say that if in a person who has been subject to migraine in early life, severe headache persists after middle life, such patient may be perfectly sound in every way, but he may very well not be so; generally speaking, indeed, he is not. A so-called migraine persisting after the age of forty-five or fifty is to be looked upon with very grave suspicion indeed. In such cases you will examine the urine very carefully, and the arteries very carefully; or, as in this case, some indications of a latent cerebral tumour may be hit upon. Mistrust "migraines," then, however "paroxysmal" the attacks may be, however typical the affection may appear, if they go on after forty-five years of age. I am almost disposed to say curtly, that if in a migrainous person the migraine persists in later life, there is probably a screw loose somewhere in his constitution.

We may now turn our attention to melancholia, mania, and other mental perversions; into these I need not enter very fully. But such cases are often very troublesome and difficult of precise diagnosis. You must search carefully to ascertain if the patient has fixed ideas, or if in certain aspects his mental habit is essentially altered. Illness of other kinds, apart from the insanities, may alter the tints of a person's character; the values of their mental features may be heightened or altered, but the character, or elements of character, is not changed fundamentally. Neurasthenia or hysteria, for instance, may make a patient more selfish, more introspective, more irritable, more extravagant, less considerate, less continent, less genial, or more lewd, than he was before, but so long as he is sane such illnesses do not convert a pure man, let us say, into a lecherous man, a man careful of money into a spendthrift, an affectionate husband or mother into one callous or indifferent. If, then, a patient enters upon a new and different character, if his previous disposition is turned upside down, we have pretty surely to do with insanity. The only exception which occurs to me is that of senile lechery first apparent with the onset of prostatic disease; and this exception is more apparent than real. Neurasthenic and many other invalids may, I repeat, become inconsiderate, whimsical, and self-centred, but I mean now something more than this; I mean that if the patient present some fresh and alien traits of character, his case is

Digitized by Google

probably one not of neurasthenia or of hysteria, but of insanity,—one of mania, perhaps, or of melancholia. This latter diagnosis becomes the more sure if you find that some idea, prepossession or obsession has become fixed, even one in which there may be some truth; if, for instance, the patient, not originally of obstinate temperament, has an extravagant son, and this fact becomes fixed on his mind to the exclusion of all degrees of comparison, of modifying conditions, and of other subjects of thought. Still more if his notion be not even founded upon fact, such as an absurd suspicion, you may be fairly sure that the case is one of no mere nervous irritability but of insanity.

I may illustrate this part of my lecture by a brief account of a very complicated case which perplexed me at times for some years; one in which no simple diagnosis was possible. The patient, a bachelor gentleman of property in the North, who had no proper occupation for a mind not by nature a very powerful one, came to me first for hysterical aphonia or dysphonia. With the help of my friend Sir Felix Semon, this malady was at length dispersed. Symptoms of neurasthenia, already present, then became more pronounced, and time and means were spent upon this state of things, again not unsuccessfully. Finally, after some interval, my patient came to me under the fixed impression that people in the streets would glance at him, and make offensive or jeering remarks upon him. He was especially convinced that he was a subject of merriment for the young women of the post-office, who, as he moved from place to place, would pass jokes about him along the wires. Surely this was rank insanity without possibility of error; yet for a time I felt by no means secure in this diagnosis. There was that in him which might well give rise to mirth; the odd cantrips of his voice, and the whimsical manners of a nervous and rather solitary man, also were noticeable enough; so, as he had boxed the ears of giggling boys in the streets, it was just conceivable that between the few places of his habitual resort some conspiracy of jokers might have arisen. And, more embarassing still, he called into my room his sister, a quiet, sensible old maid, who, even when alone with me, assured me she had certainly observed these sniggerings and the like, and believed in some widespread dissemination of chaff about her brother; a remarkable example, by the way, of the influence of mind upon mind. In order to prevent all chance of mistake, he engaged at my request a well-known solicitor to investigate the matter, and a disguised clerk was sent into certain places to pick up what was to be heard on the subject; but I need scarcely say there was nothing to be heard. In this case hysteria, neurasthenia, and insanity were mixed together in such a way as to mark well the derision of nature for our artificial, but indispensable, classifications.

Again, if a patient come to you to say, "I am losing interest in things," be alive to the presence of a very serious symptom. A mother may say, "I do not love my children or my husband;" a father may say, "I ought to love my wife and my children, and it is a misery to me that I do not." Now this may well be true; often it is but too true, the fires of love are burning low. Unlike neurotics and hypochondriacs these people lose interest even in themselves. It is no mere fretfulness, and this mental state may exist in a patient for a time with all due amiability to his family, but he comes to ask you if these "unnatural feelings" are wickedness or disease. This, again, is not neurasthenia, it is not hysteria, it is melancholia; and, if not cured in time, may mean suicide. You assure him it is disease, and you do what you can to cure him.

Hypochondriacal people are a great trial to the physician, as well as to their friends. We have to try to cure them, but the preliminary matter of diagnosis may be exceedingly difficult. To begin with, hypochondriacal symptoms are not variable. A neurasthenic person is more or less "arguable;" he may be convinced he has got some disease, but you may readily convince him that he has not a particular disease, whereupon he flies away to some other notion. He is also a very discursive person: Charcot's "l'homme à petits papiers." But a hypochondriac, on the other hand, knows the disease of his election with Calvinistic assurance; and is apt to regard a doctor who doubts his assurance as so far a fool, on whom he will waste no words. For instance, an old patient of mine, under the conviction that he was phthisical, came to me every month or so for two or three years to have his chest over-hauled; he was a vigorous, finely-built man, a good singer, and as sound as a roach. This is hypochondria, wherein the sufferer

Digitized by Google

is convinced that he has a particular disease or a particular pain; he very rarely changes his tune, and he needs no memoranda of his fluctuations, for he recognises none. A neurasthenic person may imagine for a few weeks that he has consumption; but he is glad to be reassured. He opposes no gloomy shake of the head to your words of comfort; he will harp still on his illness, but he is ready for new opinions, and even greedy of them.

Between neurasthenia and hysteria there is often no little difficulty in diagnosis. Hysteria, as you know, may occur not in women only, but in children of either sex, or in young men. Here investigation of the visual fields may give us most useful help. In England we cannot rely upon finding the curious areas of anæsthesia, which are met with very frequently in France. But if you find upon the person of a patient a patch of anæsthesia of which he was previously unaware, it is good evidence of hysteria. As regards anæsthesia in the visual field, I must utter a caution. It is usual, I think, to begin from the centre and to work towards the periphery. But asthenopic patients, such as the neurasthenical and the hysterical, soon get fatigued; and if you are taking the fields carefully and slowly, working from the centre to the periphery, by the time you reach the peripheral belts the patient becomes fatigued, his vision grows dim, and you may be led to conclude that there is contraction of the visual fields, and the disease hysteria. But give the patient ten minutes rest, and then begin from the periphery and work towards the centre, and thus you will be able to check your previous observations. In all such cases, indeed, it is better to begin from the periphery, and to work towards the centre.

In the early years of my practice I drew attention to a curious condition called the functional hemiplegia of women, now well known. I need not dwell upon the diagnosis of such a hemiplegia, especially if it occur, as is usual, on the left side. It would rarely be difficult to discriminate it from organic diseases, such as embolism, which are likely to occur in young women. It is, indeed, rather a laborious use of the limbs than a palsy; it is attended with a peculiar sense of weight in the limbs, and usually a tell-tale anæsthesia is present in greater or less degree.

Epilepsy is sometimes the cause of some embarrassment, I mean, of course, attacks of *petit mal*. Here it is of great importance to you to get accurate accounts from intelligent friends. A young man of twenty, for instance, was once brought to me by his sisters, very intelligent young women a little older than the patient, concerning some "nervous attacks." Among other curious facts they told this story, "Two or three days ago we were all at tea with people not intimately known to us. Jack, to our petrification, got up from his chair, helped himself to tea and muffin, walked off to another side of the room, and proceeded to consume it. I whispered to my sister that it was impossible that he should take such a liberty, and that he was probably in one of his 'attacks.'" I turned to the young man, and asked him "what he remembered of the circumstances?" He replied, "Well, all I can say is that it was a most distressing shock to me to find myself in two different chairs at two different parts of the room at the same moment." Now there are many inexplicable acts, many sorts of little oddities, which people ascribe for a while to "nervousness," which, in truth, are nothing less than epilepsy. And unless you have some trustworthy evidence to reason upon, or you witness an attack, it may not be possible in consultation to diagnose between such transient "turns," which are of the nature of epilepsy, and some derangement of a far less serious kind.

The subject of myalgias or muscle-pains would occupy a whole lecture, if I were competent to give it, and it would require very cautious appreciation of morbid sensations, and a sufficiency of anatomical knowledge. But, I think, if put on the track of it, if you are warned that such pains are very common and very annoying, you will succeed in your diagnosis, and thereby find some ready means of cure.

Changes of gait are often very difficult to classify. Three days ago I walked a gentleman up and down my room until both of us were tired, yet I could not make out anything morbid in his gait, although he assured me, and rightly assured me, that there was something wrong. I was strongly disposed to think it was "all fancy." He was an old gentleman, and an exceedingly neurotic one, and to my intimate knowledge had been so for many years. His friends said, "We just want you to comfort him; he says there is something wrong with his spine. Pray set his

Digitized by Google

mind at rest." Still he would repeat, "I am sure there is something wrong here," grasping his nates. Now hypochondriacal people are much given to suspicions of their nates, therefore I did not attach any grave importance to this suspicion. He begged me to "feel these muscles here." I then handled the biceps femoris and semi-membranosus, and they did indeed seem rather tight. But when I felt my own, as I stood like him a little leaning forwards, I found them rather tight too, and I got him to compare mine with his, yet he would repeat, "I do not think they are as tight as mine." As he took off his trousers I asked him which was his worse leg, and he answered that his left leg was the worse, and accordingly in it I soon detected quite definite ankle-clonus; and, to cut my description short, it became clear he had lateral sclerosis, a degeneration which had been slowly invading his spinal cord for years—for five or six years at least,—but being a highly nervous and "delicate" man nobody suspected it. I think nobody has ankle-clonus unless they have organic disease of the cord. Dr. Buzzard was, I believe, the first of us to make that statement, or to insist upon it, and the more I have seen of dubious cases, the more I have learned to rely on this opinion. At any rate it is an exceedingly important sign.*

Take another case,—a young lady under my occasional care for a long time; her sister was hysterical, or very eccentric. The patient was a good swimmer, and one day after swimming about for a dangerously long time she felt some weakness and embarrassment in her legs. When we met in consultation we could not get her even to try to walk, and no doubt there was a hysterical element in the case, but it proved to be one of disseminate sclerosis nevertheless, and of this, after a long and lingering illness, she died. Disseminate sclerosis, not inapt, as we know, to occur in young women, is sometimes a very tricky affection, and, as we have seen of lateral sclerosis, the early diagnosis may be difficult to seize. How shall we prove that this girl or boy of whimsical character has not disseminate sclerosis? What helped me in this girl's case, which I saw very early in my practice, as in the later case mentioned above, was that but a year or two before, in the Leeds Infirmary, Brown Séquard had been demonstrating ankle-clonus to me; it was then a new sign, and he told me what he thought of it as a sign of organic disease, and so on. So I tested my girl patient for ankle-clonus, and on finding it I said, "I believe Brown Séquard would say that she has organic disease." And truly, after a time slight nystagmus and slight difficulty in speech appeared; but for many months the friends were kept in very great suspense, some of us thinking the disease to be organic spinal disease with hysterical embroidery, others declaring that it was hysteria, and this alone.

Syphilis is occasionally the cause of obscure neuroses, as I imperfectly pointed out nearly thirty years ago in the 'Reports of the West Riding Asylum.' Such symptoms as melancholia, sleeplessness, and "emotionalism" may clear up quickly on a course of mercury, dictated by some such clinical hints as a doubtful eruption, a nocturnal headache, a persistent anæmia with falling of the hair, and the like.

Into the dreadful sphere of syphilophobia I will not enter, but I will venture to say a few words on the local evidence of syphilis. A few months ago I felt assured that a man under fifty years of age afflicted with arterial disease in the Addenbrooke's Hospital must have been syphilised, yet no evidence of syphilis was to be obtained. The wife had healthy children, and the man was perfectly frank and honest, saying, "I have not had syphilis; I would tell you at once if I had," Now he came at last to the post-mortem room, and I said how wishful I was to know whether he had had syphilis or not. His penis was examined, but no evidence of syphilis was found. I looked at the part myself also, and agreed that no loss of substance was apparent. Then came in a well-known Cambridge surgeon, to whom I said, "We are inquiring if this man has had syphilis." He took up the penis, looked at it carefully, retracting the foreskin, and said, "No, I do not think there is local evidence of it." Afterwards he returned, saying, "That was not a thorough examination;

* Yet a very remarkable case of recovery in a patient who had presented ankle-clonus, is vividly in my memory. Some years (five or six) ago a middle-aged woman came into the Addenbrooke's Hospital with symptoms of lateral sclerosis. She was unable to walk. Ankle-clonus was well marked in both legs. She was discharged without relief. A few weeks ago she was readmitted for bronchitis, and her paralytic symptoms and her ankle-clonus had wholly disappeared. She "gradually got well."

Digitized by Google

let us try again." Again he pulled the foreskin back, and, taking a sponge, he cleared all the smegma and such matter away from it, when very near the frænum, and not so large as the sectional area of a split pea, was a circular white, slightly depressed, silky scar without a pucker upon it: absolutely unmistakable. The man had never known of his chancre, and secondary symptoms, if any, had been slight and transient. This case shows how much value to attach to negative post-mortem records, or even to clinical evidence for that matter.

I cannot go very exhaustively into the diagnosis of latent syphilitic maladies, suggesting "neuroses." The eyes, of course, offer a very important standard, though I must speak with diffidence in the presence of our chairman, Mr. Hutchinson. If you find the pupils unequal, and the light-stop in the iris, which is alleged to be evidence of syphilis,—*i. e.* reaction to accommodation, but no reaction to light, remains of old iritis or corneitis, and so forth—evidence of syphilis is grave. The optic fundus, again, may contain evidence, either positive or negative, of great value; such as optic-nerve disease, retinitis, or choroido-retinitis. Pegged teeth will not be overlooked.

Perhaps the most grievous blunders in diagnosis, blunders which are more than possible, would be to confound "Landry's palsy,"—using the name in a broad sense—or, again, that disease which has more recently come under stricter observation—the so-called "Myasthenia gravis," with functional neuroses. I have recorded an instance in my own practice, when provisionally, and with some hesitation, a girl was supposed by some of us to be the subject of hysteria, who died suddenly a few weeks later from myasthenia gravis. Such errors are much to be lamented, however impossible in some cases it may be to avoid them. If we remember that such precipitous issues are to be reckoned with, we shall be on our guard lest we misread the signs of them. I may travel outside my subject so far as to compare such cases with those no less distressing, in which young women, rightly regarded as but chlorotic, die as suddenly of thrombosis of the cerebral sinuses. I well recall three such accidents, but, at present at any rate, we have no means of foreseeing them.

I may now consider the diagnosis between functional perturbations and diseases of the heart or arteries. Some of these cases, seemingly "neurotic," are organic. A person of some fifty years of age may come to you, in whom you will find it very hard to say whether his discomforts are but "functional" or "organic." Perhaps he smokes a good deal, or drinks much strong coffee, or is worried or overwrought, or, on the other hand, he may be the subject of some graver disease. On the contrary, there are cases of very grave aspect, even cases of angina pectoris, which end in recovery. I have seen more than one instance of men under heavy mental stress who have presented symptoms which were not to be distinguished from angina pectoris, which, indeed, were clinically angina pectoris, but who in the course of a year or two got well nevertheless. Whether in such cases aortitis had been present I cannot tell; aortitis may arise from some such toxic cause as influenza or syphilis, and pass away. In comparatively young men who have the time and the means for long and careful treatment, rest, and climatic change, do not despair even of angina pectoris, unless the morbid state of heart or arteries obviously forbid your hopes.

Again, to turn to another aspect of arterial disease, a gentleman was brought to me not very long ago by a medical practitioner under whose care he had just come, after seeing five or six medical men in consultation and otherwise, who regarded the case as one of neurasthenia, and such, indeed, the symptoms seemed to be. We examined the case together. He was a man of some forty-five years of age, a stockbrocker, who had, no doubt, "done himself very well." On this visit we found the apex of his heart fully up to the nipple line, and even a little beyond it; and the quality of the impulse was distinctly heaving. There were no murmurs, but the aortic second sound was very loud. The radial pulse also betrayed a very high arterial blood-pressure. While we led him to hope for a good recovery, we told him his case was not one of neurasthenia at all, but a disordered state of the circulation, chiefly conspicuous in the arterial system. Such cases as these end in arterial degeneration if not promptly and persistently treated, as I have described at length on several occasions.

Let me now discuss the difficulty of diagnosis in those not rare cases of Graves' disease, in which no eye symptoms are to be noted, whether von

Digitized by Google

Graafe's sign or Stellwag's sign, or proptosis, and also without palpable enlargement of the thyroid. In such cases the quick pulse, a pulse say of 110, 120, or 130, with arterial atony persisting for months, and, indeed, years, attended with debility and emaciation, and yet without any other very definite symptoms may stand alone; though diarrhœa and vomiting, or a fine tremor may be present to guide us to a decision. We shall not forget, then, that mere quick heart, standing alone, is not rarely Graves' disease. If on the other hand the quickening of the heart be not continual but paroxysmal, the malady is probably that known as Paroxysmal tachycardia, a disease which has now been described so clearly as to command our attention, and to ensure a right diagnosis. There are, however, various drugs, such as coffee and tobacco, whose effects may simulate larval Graves' disease, or some form of neurasthenia.

With regard to nephralgia, it is alleged that women or men without stone in the kidney, but with intense nephralgia, may be attacked with hæmaturia. I do not know whether this be true or not, but so it is stated. A careful examination of the urine in the intervals between the attacks may lead us to a diagnosis; and if there be no hæmaturia the diagnosis between "nephralgia" and renal colic is rarely very difficult. A greater difficulty often lies in the distinction between gastralgia and ulcer of the stomach. In neurotic young women we are apt to say that gastric pains are but nervous, and that no lesion is present, whereas the case may be ulcer of the stomach all the while. Or, again, symptoms simulating those of neurasthenia may be due to moderate degrees of dilatation of the stomach after diseases like influenza or acute rheumatism, for example; or to dilatation due to obstruction, or, again, to the condition called enteroptosis, when the stomach may drag upon the pyloric attachments and kink. A gentleman once came to me who for years had been regarded as neurasthenic, fanciful, or idle. I found in him a dilated and catarrhal stomach, the proper treatment of which, when I last heard of him, had given him considerable relief. In such cases the symptoms due to the toxic absorption—such as incapacity for exertion of mind or body, hours of torpor, or of fretfulness, and the like, usually betray themselves by their intermittency; and physical examination should settle the matter. A gentleman of the age of 35, a schoolmaster, once came to me suffering thus from intermittent nervous symptoms and disabilities, which I found to depend with curious regularity upon recurrent and most obstinate states of constipation. By minute attention to the bowels the general symptoms were got rid of.

Here I may bring before you those painful cases of intermittent, or even of persistent, pain due to adhesions of the stomach to the wall of the abdomen. In the early years of my practice such a case came before me, and was regarded as one of hypochondriasis, or of neuralgia. The patient, a very cultivated and useful man, had his life rendered almost ineffectual by "gastralgia," and some subjection to the habit of morphia was almost necessarily entailed upon him by the recurring epigastric pain. Those were not the days of ready laparotomy; but he desired that at his death a necropsy should be made, when the cause of the pain was found to be a short fibrous cord running from the stomach to the anterior wall of the abdomen. I have known definitely of two such cases since, and have surmised the presence of such a condition in others. A history of ulcer or of peritonitis might aid us in interpreting pain due to such a cause, and direct our attention to surgical exploration.

There are various urinary disorders, such as baruria, oxaluria, and lithiasis, which may give rise to nervous symptoms, or be attended with them; but no doubt everyone nowadays examines the urine as a matter of routine, and I ought not therefore to suggest these cases as offering any difficulty of diagnosis.

The last difficulty I shall touch upon is that of covert malignant disease, a dreadful alternative. A feeble and wasted mortal, of cachectic aspect, aged, say, fifty-five or sixty-five, comes to you for advice with neurotic symptoms enough. But is it all "neurosis?" He may have lost his flesh of late very rapidly. You overhaul him from head to foot, but you may find no local mischief. Still the dark suspicion remains, as malignant disease may be hidden somewhere or other nevertheless? Even after long consideration one can but say to the patient, "I have been carefully over the different parts of your body, and I find nothing. I cannot be sure, however, that you have no malignant disease until I have put you through the Weir

Digitized by Google

Mitchell treatment, and observed its results." Still, taking history, symptoms, and physical examination together, we should attain, and after a certain amount of experience we do attain, to the art even of negative diagnosis, and usually find ourselves able to tell the patient pretty confidently that he has not malignant disease, but neurasthenia. Yet negative diagnosis has its lesser as well as its greater risks of error. For instance, I went two or three years ago to see a gentleman of advanced age, a healthy old country gentleman but of very nervous temperament, who impressed upon us his conviction that he was the victim of malignant disease of the rectum. He drew our attention especially to his discomfort from bearing down sensations and his desire to empty the bowel. Everybody, lay and medical, had told him it was all nonsense. But when I came to make a rectal examination, I convinced myself of the presence at that moment of something abnormal just within reach of the tip of my finger. It was a soft and velvety enlargement of some kind ; not like a malignant growth, but rather like a mucous sac containing some fluid. My medical friend then passed in his finger, and said that he also certainly felt something, which he agreed with me was not itself malignant disease, and probably not even associated with such disease. This opinion we gave to the patient himself, who soon found a surer consolation. We told him to watch his motions, not using the water-closet, and a week or two later the swelling burst. It turned out to be a submucous sac which had become distended with chylous fluid. After the bursting of the cyst he became quite comfortable, the cyst refilled two or three times when the disorder came to an end. All this must have happened three or four years ago, and I know that his health was good three months ago.

In conclusion, my only excuse for so long an occupation of your time is my desire to help you to arrive at opinions and to find definite lines of treatment in a class of cases of much obscurity, cases in many of which what may be called a negative diagnosis may be the right one ; but to rely upon a negative diagnosis is often a very trying thing to have to do. If I have said anything which will give you a clue to the direction in which hidden defects or dangers may lie, and the devices by which, in the midst of a fog of neurotic complaints, you may detect local disorders, which either simulate mere neurosis, or aid and abet it, I may have done you some little service. To discriminate between nervous troubles and local diseases requires great tact and experience on the part of the medical man, and yet without such discrimination he may fail in his mission of cure. It will not do to dismiss patients in whose body no signs of a definite and significant character can be discovered—that is to say, no abdominal tumour, no cardiac tumour, no pulmonary mischief, no albumin—on the ground of mere "nervousness." Such rough and ready decisions I often call "lumps and murmurs diagnoses." No lump, no murmur, no albumin—no disease. You will agree with me that to practise medicine successfully we must carry diagnosis to a higher degree of refinement than this.

F. W. Tunnicliffe and O. Rosenheim ('Journal of Hygiene,' April, 1901; 'Centralbl. f. Physiolog.', April 27th, 1901), have made experiments by feeding apparently healthy and delicate infants with articles containing boric acid or borax, and made careful analytical and other observations as to the effects on nutrition and metabolism. They arrive at the following conclusions, which are not likely to strengthen the hands of those who wish to restrict the use of such agents. Boric acid in small doses up to one gram per day produces in healthy or delicate children no change in proteid metabolism and no appreciable influence on the assimilation of fat. The body-weight is increased. Similarly, borax in 1·5 grams doses per day produces no unfavourable influence on proteid metabolism or assimilation of fat, and the body-weight is increased. The amount of dry feces is slightly increased with borax, but not with boric acid. A mixture of boric acid and borax (which has been found to be a better preservative than either alone) is quickly eliminated and does not act unfavourably. Concerning formaldehyde, the observers find as follows: Formaldehyde added to milk in the proportion of 1 to 5000, or to the entire aliment in proportion of 1 to 9000, has no appreciable influence upon the more important phases of healthy children, but with debilitated children some interference is shown. In no case was any unfavourable influence upon the general health of the children noticed.—*Philadelphia Medical Journal.*

Digitized by Google

A CLINICAL LECTURE

ON

CHOREA OR CEREBRAL RHEUMATISM.

Delivered at St. Bartholomew's Hospital, May 24th, 1901,

By Sir DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Physician, and Lecturer on Clinical Medicine, St. Bartholomew's Hospital.

GENTLEMEN,—I propose to discuss with you to-day the case of a girl, A. M—, æt. 12 years, who was admitted into John Ward on April 25th, 1901, suffering from chorea in a severe form. You know that this is a disease characterised by irregular involuntary movements of all parts of the body, met with chiefly in youth, and in the female sex. It is more often seen in the children of the poor, though minor phases of it are met with in all classes of society. When you have once studied the features of a case of this disorder you will readily recognise them again. I bid you take note of the fact that you may meet with the disease in varying degrees of severity. The gravest cases are very distressing to witness, and these occur commonly at the age of puberty.

This child is fair-haired and chubby, well grown, and with large blue eyes. She had been in good health up to four months ago, when she had a severe sore throat, since which time she has been languid, and had a poor appetite. A week before admission she had pains in the left hip and thigh, and limped. Later, she had severe pain in the left wrist. Her speech became stuttering and "clipped" at this time. Three days before admission fidgety movements set in in the arm and legs, more especially on the right side. The child had been recently working for an examination at school. I note this point, because there is no other evidence of any special shock or emotion to be obtained in the history. The previous health had been good, with the exception of measles and whooping-cough some time back. We learned that the mother once suffered from chorea, at the age of twelve, and was the subject of heart disease. There are two brothers and four sisters living and well. No other history of rheumatism in the family.

I pause here to remark that, had we had to deal with the early ailments in this case, we should undoubtedly have pronounced them of a truly infective rheumatic nature. The sore throat was almost certainly rheumatic, and so was the arthritis which succeeded it. We find, then, supervening on these rheumatisms of the throat and joints the occurrence of these choreic manifestations. That is the order of events. The child was extremely restless, and had violent irregular jerking movements of the body and limbs, her face being hideously distorted by grimaces. She could not speak, and the tongue was with difficulty thrust out momentarily after attempts to protrude it. The respiration was jerky and irregular, 26 per minute. The whole aspect was that of maniacal excitement, and to such cases the term *chorea insaniens* is therefore applied. The disorder rapidly increased in severity, and we had to arrange a padded bed, with sides to it, to prevent injury and falling out. The temperature was febrile on admission, 100·2°. I hand round the chart made up to to-day. The heart's apex-beat was in the normal place, the cardiac dulness natural, and the sounds clear, and of good tone. It was evident, then, that the rheumatic poison had not involved, as it so often does, the endocardium. The chest and abdomen were natural, and the urine was of rather high specific gravity, but free from albumen or sugar. The knee-reflexes could not be ascertained owing to movements. Milk diet was ordered, with Mellin's food, and gr. iij of calomel were given. Ten grains of salicylate of sodium, with five of potassium bromide were given every fourth hour. The bed was screened off so as to keep the patient as quiet as possible.

The main object in these cases is to secure as much sleep as possible, and to attend to the feeding, the disorder being an exhausting one owing to the constant movements. Chloral hydrate is one of the best drugs to employ, the only objection to it being its tendency to depress the action of the heart. We therefore employed it in doses of ten, and then of fifteen, grains given every six hours. It proved of little avail, however, and as the pulse became weak we prescribed paraldehyde in drachm doses every six hours. This proved of much use, and secured some sleep and diminution of the active movements. In the progress of the case nothing of moment occurred,

Digitized by Google

but the disorder continued severely till the 2nd of May. The paraldehyde was suspended for a time, and sulphonal and trional were given. Nasal feeding had to be resorted to in order to secure a due supply of nourishment. Ether spray was applied to the spine, but did not appear to be helpful. The diet was improved on the 5th of May, and some hypodermic injections of hydrobromate of hyoscine were employed on three occasions, $\frac{1}{100}$ and $\frac{1}{75}$ of a grain being given. The bromide and salicylate draught was resumed; also paraldehyde, latterly in drachm and a half doses. This was kept up for some days, and next we tried antipyrin in ten-grain doses every six hours, paraldehyde being still given at intervals. Not till the 18th of May did any decided improvement occur, and from that time till the present there has been amendment. The heart's action has been well maintained, and no murmurs have been noted. We may now trust that the activity of the disorder has been subdued. I show you this patient, and you see that there are still irregular movements present. Silence has been marked all through the illness, but consciousness has apparently been clear. The girl is very emotional and excitable. I confess I feared this was likely to prove a fatal case, and but for the sedative remedies steadily employed, and the assiduous nursing day and night, I still think we should have lost this patient. It has been alleged that fewer cases of severe chorea die nowadays than formerly, and that life is saved by means of chloral hydrate medication, and I believe that this is the case.

For some years past, there has been a growing opinion to the effect that chorea is a malady dependent on the entry into the system of some toxin. The close association of rheumatic manifestations has long been noted. One of the latest English writers on the disease declares that "the only hypothesis which will satisfactorily associate the functional disturbance of the nerve-centres and the very manifest and tangible endocarditis, is the view that they are both the result of some poison or virus, or toxin circulating in the body. This case is, however, not singular in showing no sign of endocarditis. In fatal cases, it is, however, rare not to find signs of active endocarditis, and in many instances it is to be met with during life.

To my mind, the association with rheumatic manifestations is of supreme interest and importance. Statistics prove the great frequency of this association both in family and personal history. It is clear enough in this particular case. Endocarditis is not solely the result of rheumatic influence, as you well know. Many infectious disorders may induce this condition. The writer just quoted —my friend, Dr. Taylor, of Guy's Hospital—is of opinion that the occurrence of endocarditis "does not for one moment prove a connection with rheumatism, since there are other causes of endocarditis." I join issue with him. For twenty years I have taught that the majority of cases of chorea are neither more nor less than *rheumatism of the brain*. In 1894 I read a paper in Rome at the International Congress, urging this view, and a further study of the matter has more fully convinced me of the truth of it. It is a narrow and vulgar view to take that rheumatism is a disorder only manifested by pains and swellings in the joints. We certainly meet with rheumatism of the throat, of the skin, of the muscles and fascia, as well as of the joints, and so there are many rheumatisms affecting varied lines of tissue. And, depend upon it, there is cerebral rheumatism, and that is manifested by chorea. Infective disorders, of which rheumatism is one, are always prone to alight upon points of least resistance, upon weakened or over-wrought structures. In many cases of chorea, we not only find history, family or personal, of rheumatic disorders, but we also find that there is a feeble or unstable cerebro-spinal system, a neurotic predisposition, we find that there has been over-excitement, shock, fright, or emotion, over-strain in study, or brain pressure, rendering the encephalon liable to break down or to be involved in any invading infective process. And so if any infective toxin gains access to the body at such a time, its manifestations are prone to appear on this line of exhausted or weakened tissue.* We do not, as a matter of fact, often meet with chorea in association with any other infective disorder but rheumatism. Other antecedents have been noted, as influenza, measles, scarlet fever, varicella, and typhoid fever. These as immediate antecedents are most rare in my experience. No

* All persons are not equally prone to suffer from rheumatic infection. This liability distinctly pertains to a certain but wide-spread habit of body, which provides a suitable soil for its manifestation.

Digitized by Google

pathologist can tell in the post-mortem room of any difference whatever between the endocarditis of chorea and that of rheumatic fever. The two conditions are exactly alike, and for the best of reasons, as I think—because they are identical, and the result of the same toxin in each case. And now we proceed a little further, and come to consider some recent researches which add support to my contention. First, let me say that I have witnessed the onset of chorea during an attack of rheumatic fever, and that rheumatic arthritis may occur in the course of cases which have been purely choreic from the outset. There is no going back on these facts. At this moment you will find a young female patient in Hope Ward, under Dr. Gee's care, who was lately admitted with large patches of erythema marginatum on the trunk—a variety, that is, of rheumatic erythema,—and who has developed chorea since she came into the hospital. Is this association of no clinical significance?

In their recent able and instructive researches into the nature of rheumatic fever by Drs. Poynton and Paine,* it has been demonstrated that the intravenous injection of the specific diplococci led to choreic symptoms in a rabbit. On subsequent inspection the diplococci were found in the pia mater, and endothelial cells and walls of the capillaries. The pia mater showed slight swelling of its connective tissue and cell-exudation. The same animal had endocarditis and polyarthritis. In another rabbit paresis of the limbs occurred, suggestive of the paretic condition which often ensues in the limbs of human subjects of chorea. In a case of fatal chorea in the human subject, the micrococci were found on the mitral valve and in the motor centre. As these organisms clearly gain access to the blood-stream, it is easy to realise that they must reach the cerebral circulation, and may thus escape into the brain substance.

These observers suggest that the early symptoms of chorea depend on the presence of diplococci in the brain or pia mater, and regard it as likely that morbid changes ensue of analogous character to those met with in other parts, on which these organisms alight. These changes consist of swelling, hyaline degeneration, and sometimes of necrosis, with connective tissue proliferation, hyperæmia, and hæmorrhage. Some of these are probably due to the toxins generated by the organisms. Half a century ago, Dr. Kirkes, of this hospital, suggested that chorea was probably induced by particles embolically shed from the inflamed endocardium into the brain motor centres. This was the so-called "embolic theory" of chorea, which held the field for some years, supported by many authorities. In some cases the emboli were discovered; but in many they were not detected, and then the theory was held to be inadequate to explain all cases of the disease, and so was discarded. Bacteriology was then in the womb of the future. We have now better means of pursuing the necessary inquiries relating to small, and even infinitely small yet particulate, fragments which may be embolically shed into the finest vessels, impregnated with infective and specific quality.

By the light of these fresh researches I think we see our way more clearly than before to realise that in chorea we are in face of a cerebral disorder determined by an infective organism, and that organism is the specific microbe of true rheumatism. Careful studies of the brain in fatal cases, made in recent years, have shown that hyperæmia of parts, softening from minute emboli, perivascular exudations, swelling of nerve-cells, and lepto-meningitis may be met with, and these are now readily explicable as results of this infective process. We often meet with symptoms in chorea which indicate the localization or severer manifestations of the brain disorder. Thus we find hemichorea, or the predominance of symptoms on one side. The speech centre appears to be commonly involved. It is so in the patient before us, and we have noted that her right limbs are specially affected. In such a severe case, if we bear in mind the possible damage wrought in the brain, we must expect that recovery will be slow, and that many weeks must elapse before the centres resume their proper functions. In my next clinical lecture I propose to continue this subject and to discuss the predisposing conditions relating to cerebral rheumatism, the incidence of it, and the various therapeutical indications which determine the line of treatment for patients suffering from chorea.

* 'Lancet,' p. 1260, May 4th, 1901.

Digitized by Google

CLINICAL REMARKS UPON THE TREATMENT OF THROMBOSED VARICOSE VEINS OF THE LEG.

By C. MANSELL MOULLIN, M.D.Oxon., F.R.C.S.,

Surgeon, and Lecturer on Surgery, at the London Hospital.

THE question, gentlemen, to which I wish to invite your attention to-day is the line of treatment that we should adopt in cases of thrombosis of the superficial varicose veins of the leg, of which we have recently had a large number of cases in our wards. The condition is a very common one, not only among hospital patients, but in private. It is especially frequent in that part of the internal saphena that lies by the side of the knee and the lower third of the thigh, though it is by no means confined to this, and it is undoubtedly a condition in which there is a grave degree of danger. Only a short time ago a patient who was in one of our wards waiting for operation died suddenly from a portion of the thrombus becoming detached and carried into the circulation; and such accidents are by no means rare.

I need not trouble you with the question of diagnosis. You cannot mistake for anything else the soft purplish masses along the course of the vein, standing out underneath the skin and more or less adherent to it. There is no complaint which resembles it, or is likely to be mistaken for it. But as there is always a certain amount of inflammation around a thrombosed vein, a certain degree of tenderness, redness of skin, and swelling of the surrounding cellular tissue, it is advisable before coming to any decision as to what you are going to do to note carefully how far this spreads. Every now and then you meet with a case of inflammation of the cellular tissue which spreads into and involves the walls of the veins, in which the phlebitis is secondary to infective inflammation around; and these cases must be clearly distinguished from those of which I am speaking now. I wish to confine myself to-day entirely to those cases in which the thrombosis is the prominent feature, and the inflammation around secondary to it, and comparatively slight.

A word or two first about the causes which give rise to this thrombosis. They are three in the main and are fairly clear, though you will often find some difficulty in determining the relative share they take in individual cases. Slowness of the blood-stream is one, and for this reason you nearly always find that the thrombus starts from a dilatation, in which presumably the blood is almost stagnant, or from behind a valve. Impaired vitality of the endothelial lining of the vein is another. Sometimes this may arise from prolonged stasis, the nutrition of the lining cells slowly growing more and more feeble, until at last some fibrin ferment is set free; but much more often it is due to actual injury, a blow, or the compression of the sharp edge of a bandage. You will very often hear that the thrombus began to form after the receipt of some blow or sudden strain. The third, the value of which it is much more difficult to estimate, is some alteration in the constitution of the blood itself. In one of the cases upon which I operated recently, if you remember, the whole length of the internal saphena on both sides was thrombosed from below the knee up to the saphenous opening, a condition which clearly could not have arisen from a mere local cause acting by itself. But what this alteration in the constitution of the blood may be is by no means easy to say. Probably it is not always the same. In all wasting diseases, such as phthisis, you are liable to get thrombosis. In severe cases of typhoid it is quite common, and it not unfrequently occurs after grave operations, even when the wound pursues a perfectly aseptic course, setting in very often ten days, and even three weeks after, when the patient is almost convalescent. To avoid this you will see that the lower legs of the beds in our wards, after severe and especially abdominal operations, are raised nine and even twelve inches on wooden blocks. This tends to prevent venous stasis, and so diminishes the chances of a clot forming. Probably in many of these cases bacteria play a not unimportant part. They enter into the circulation, as they easily can, but are not sufficient either in power or in number, or in both, to cause any general effect. Only where the blood is circulating slowly and feebly, or the wall of a vein has been injured by a blow or the prolonged compression of a bandage, they have just power enough to cause the formation of a thrombus. This at least has been proved to occur when the bacillus coli or the

Digitized by Google

typhoid bacillus is introduced into the circulation in animals under somewhat similar conditions, and it is therefore highly probable that it takes place in man as well. If it does, it will serve to explain a good many of those instances in which the sudden occurrence of a thrombus has often appeared to be rather mysterious. I do not like to push an argument of this kind too far, but it is certainly significant that thrombosis of the veins in the lower extremities is particularly liable to occur after abdominal operations, in which the bacillus coli is not uncommonly present.

Now, gentlemen, what is to be done with patients suffering from this complaint? The routine treatment is to keep them perfectly quiet in bed, with the affected limb covered up either with lead lotion or extract of belladonna according to the severity of the inflammation that accompanies the thrombosis, and wait until absorption and organisation are complete, until all pain and tenderness have gone, and the vein either has regained its natural condition or feels hard and cord-like. In the meanwhile the patient is liable to sudden embolism, which may occur at any moment, and to extension of the thrombus, until either it spreads through the saphenous opening into the common femoral, or what is nearly as serious, extends through some of the communicating branches into the superficial or the deep. And if you think for a moment of the condition that is actually present—a blood clot, perhaps the size of a large cherry, lying in a portion of a dilated vein, surrounded by a layer of passive endothelial cells resting upon a comparatively non-vascular basis, you may perhaps be able to form some idea of the length of time that absorption and organisation may take. It is always weeks. It may be months, and I have known it longer than a year, during the whole of which time the patient was kept lying in bed, leading an almost useless life, and living in a constant state of apprehension, never sure that something might not happen.

Seeing how unsatisfactory this is, for some years past I have adopted a totally different course. My first case was more than ten years ago. The patient was a gentleman who, while standing on tip-toe trying to reach something from a shelf high up over his head, was suddenly seized with a sharp pain on the inner side of the thigh. On examination, there was a characteristic thrombus in a dilated varicose vein. Owing to circumstances, it was impossible for him to lie up for any length of time, so I made an incision over the vein, turned the thrombus out, tied the vein above and below, and sutured the wound. Within a week he was perfectly well, and suffered no further trouble.

Since then I have adopted this method in a large number of cases. I prefer to put a ligature first around the vein immediately below the saphenous opening, to prevent any of the clot, if it is accidentally displaced during the operation, being carried off as an embolus; but in several instances, as you have seen, the internal saphena vein was blocked quite up to its end, and it was essential to withdraw the clot from its termination before this could be done. Then, if possible, all the thrombosed parts below are dissected out; or if this cannot be done owing to the length of the vein involved, portions here and there are removed; the clot is cleared out from the intervening segments; and the ends of the vein left behind are closed either by ligature, or, better still, by torsion, as then no foreign substance is left in the tissues. In some of those cases in which there is a large grape-like mass on the inner side of the thigh, and especially in those in which the wall of the dilated vein is more or less adherent to the surrounding structures, so that it cannot be readily separated, I have adopted a more summary measure still. I have merely reflected a flap of skin from off the vein, slit the tortuous masses up in all directions until the clot could be turned out completely, removed such portions of the vein as would come away easily, and then replaced the skin.

In cases such as these I cannot speak too highly of this method of procedure. It is perfectly safe, provided proper precautions are taken. The redness of the skin, the tenderness, and all the other signs of inflammation disappear at once. Union takes place within the week. The patient is able to get up and go about his work before a fortnight has passed. He is freed at once from all risk of embolism, and the cure is final. The only drawback is that the use of this method must be restricted to the superficial vessels. When the thrombus involves the femoral vein, whether it originates in it, or spreads into it, there is nothing for it but unlimited time and patience.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY

No. 458. WEDNESDAY, AUGUST 7, 1901. Vol. XVIII. No. 16.

CONTENTS.

PAGE

*A Clinical Lecture on Certain Complications occurring after Operations for the Cure of Hernia. By SIR WILLIAM H. BENNETT, K.C.V.O. ... 241

*Two Clinical Lectures on Intestinal Ulceration. By C. R. B. KEETLEY, F.R.C.S. LECTURE I. 247

*Some Remarks on the After-effects of Anæsthetics. By J. BLUMFELD, M.D.Cantab. 252

Treatment of Fractures. JOHN POLAND, F.R.C.S. 256

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

CERTAIN COMPLICATIONS OCCURRING AFTER OPERATIONS FOR THE CURE OF HERNIA.

Delivered at St. George's Hospital.

By SIR WILLIAM H. BENNETT, K.C.V.O.,

Senior Surgeon to St. George's Hospital; Member of the Council of Examiners, Royal College of Surgeons, etc.

GENTLEMEN,—As I have often had occasion to say before in these lectures, the most useful clinical lessons, for those who will take the trouble to learn them, are provided by common cases, and not by rare ones. There is now in the Hunter Ward a man who has been suffering from a very common complaint, in the form of an irreducible omental inguinal hernia, upon whom I operated a short time since with a view to bringing about a radical cure. He is fifty-five years of age, an Irish labourer, who has consumed a great deal of beer in his time, but still is, for all ordinary purposes, a healthy man. The hernia was on the right side, and a very large one, hanging almost halfway to his knees, and it was, as I have already said, irreducible. The case was, so far as local conditions were concerned, as unfavourable a case as could, under ordinary circumstances, well occur for the performance of the radical cure. At the same time no truss, nor any other appliance which the man had been able to use—and he had tried many—gave him any help, although the pressure of some of the trusses used had been so great that ulceration on the skin surface of the hernia followed their use. Under the circumstances, therefore, in spite of the man being an unfavourable subject, and in spite of the hernia being of an unpromising kind for the radical cure, it was decided that the operation should be attempted. At the operation there was found in the sac, as some of you may recollect, an enormous mass of omentum, more than a pound in weight, in addition to which there were several feet of small intestine. The small

Digitized by Google

intestine was, of course, reduced in the ordinary way; and then came the question as to what was to be done with the omentum. The omentum in the sac was really the whole of that structure, greatly increased in bulk from overgrowth. The transverse colon, to which this omentum was, of course, attached, was dragged down by it in the form of a loop into the neck of the sac. It was clearly necessary that this altered omentum should be removed. A rather delicate question then arose—as it always does arise in cases of this kind,—about the best method to get rid of the superabundant omentum. Would it be better to pull down the omentum further into the sac, in order to place the ligatures upon the healthy or natural portion of it; or was it better, under the circumstances, to apply the ligatures about the hypertrophied and overgrown part of it which lay in the sac? For a reason which I shall presently mention I thought it better to pass the ligatures, before cutting away the omentum, through the proximal part of the hypertrophied portion of this structure, as it lay in the sac. A number of silk ligatures were therefore applied, so that after the removal of the ligatured mass four considerable stumps were left; these were returned into the belly, and the operation completed in the usual way. Nothing could have been more satisfactory than the subsequent progress of the patient. The wound healed immediately; the bowels acted on the third or fourth day, and everything seemed to be going on perfectly. At the end of ten days he was lying comfortably in bed, but feeling somewhat irritated at not being allowed to get up. On the fourteenth day after the operation, the action of the bowels being regular and free, without warning of any kind, he suddenly had an attack of acute pain in his belly, and vomited profusely. This vomiting recurred several times, the pain became more intense, and the belly became swollen and a little full. In addition, great difficulty about getting the bowels to act supervened. In point of fact, all the cardinal symptoms of intestinal obstruction came on—sudden pain in the belly, vomiting, increasing distension, and inveterate constipation. I saw the man about twelve hours after these symptoms had supervened, because Mr. Mummery, my house surgeon, thought that the case was presenting rather a threatening aspect, which was perfectly true. I saw the patient and examined his belly, and found him in the condition I have mentioned; but I at once came to the conclusion that there was no necessity for any further operative interference, nor, indeed, for great anxiety, because, although the symptoms were so very like those of intestinal obstruction, as a matter of experience I knew they were very unlikely to be associated with that condition at all. With a little perseverance we got the bowels to act, and although vomiting recurred at intervals for several days, by degrees the pain subsided, the distension of the belly went down, the patient soon recovered, and is now in a comfortable and happy condition.

This is a very instructive case, and the question arises, what was the cause of this extraordinary recurrence of symptoms? You will remember I said that at the time of the operation large masses of omentum were ligatured, in fact we removed nearly two pounds of omentum after having ligatured the proximal part of it by passing ligatures through the hypertrophied and altered portion. It is a singular thing that after operations for hernia in which the omentum is interfered with, recurrent symptoms strangely like those of intestinal obstruction are apt to occur. These occur at two periods: either immediately after the operation, that is to say within the first twenty-four or thirty-six hours, or from the tenth to the fourteenth days subsequent to the operation. This case of which I am speaking belonged to the second class.

I will now give you an example to show what the other class of case is like. A man aged twenty-eight had a hernia in the groin, the cure of which was attempted by means of the radical operation. A piece of omentum the size of two walnuts was in the sac, and it presented a lobulated appearance. This was removed, and in order to remove it the omentum was pulled down out of the abdomen until a healthy unaltered portion was arrived at, around which the ligatures were applied—the best plan under ordinary circumstances. The wound was sewn up, and the operation completed in the ordinary way. The patient was quite comfortable the same evening. The next morning he was very ill; he vomited constantly, had a temperature of 102°, his abdomen was becoming distended, and he was in acute pain. His face had a flushed uncomfortable look, and the case altogether assumed a very

Digitized by Google

threatening aspect—the recurrent symptoms having come on almost immediately after the operation. They were symptoms strongly suggestive of peritonitis at all events, if of nothing worse. Judging by the appearance I have had in similar cases, in spite of the threatening aspect of the case, I did not think very gravely of it. My impression was that in the course of twelve or twenty-four hours the symptoms would subside, the vomiting would cease, and that the patient would then get along well. All therefore that I did was to give him a hypodermic injection of morphia to make him as comfortable as possible, and to ensure free evacuation of the bowel by enemata and repeated small doses of salines. The result was very much as I had anticipated. In thirty-six hours from the time these symptoms came on the patient was comfortable, and the case pursued a clear and easy course.

In this case you will notice that the application of the ligature to the omentum was not around the altered omentum, but around healthy omentum which had been pulled down out of the abdominal cavity in order to provide a stump for the ligature. The altered portion of the omentum was cut away on the distal side.

These two cases are very useful for contrast. Both were cases of omental hernia ; in both cases symptoms apparently of a very serious kind occurred after operation, and in each case, in spite of the apparent gravity of the symptoms, I declined to interfere further and ventured to give a very favourable prognosis. This was at first sight a heavy responsibility to incur, because in the first case there was apparently a strong probability of intestinal obstruction. and in the second case it appeared very likely that there might be acute peritonitis or something of that sort coming on. The negative attitude which I assumed in both these cases, and always do assume in such circumstances—I have seen very many of these cases,—was dependent really upon the fact that the parts dealt with in the operation were omentum only. Supposing that in either of these cases the parts involved in the hernia had been gut only, and more especially if the gut had been strangulated, I should have made in the first case an abdominal exploration, and in the second instance I should have opened up the wound to see what was going on inside the belly. The difference in the gravity of symptoms like those I have described arising after operation for hernia containing (1) omentum only and (2) gut only is of great importance and interest.

Let me impress upon you the following interesting fact:—threatening symptoms coming on after operations for hernia in which omentum has been dealt with by ligature and removal, may be looked upon as being most likely of not much importance, provided the case is managed properly. On the other hand, if symptoms of a similar kind come on in a case in which gut only has been dealt with, they are much more serious, and may need abdominal exploration.

Why is it that these recurrent symptoms are so prone to occur in cases of omental hernia. It would naturally be thought as a matter of course, at first sight, that if a case of strangulated hernia had been recently operated upon in which gut had been alone involved, that the mere fact of the gut being a more important structure for ordinary surgical and physiological purposes than the omentum, untoward symptoms would be more likely to arise in a case of that kind than in one in which omentum had only been concerned. But in point of fact the reverse is the case. Symptoms of a threatening kind after ligature of healthy omentum in dealing with these cases of hernia are not at all uncommon—symptoms such as vomiting, pain, and high temperature, within twenty-four or thirty-six hours ; or at a later period, as in the first case I have spoken of, twelve or fourteen days afterwards, or even later than that when the ligatures have been applied round about old insensitive hypertrophied omentum. The reason of the tendency to the occurrence of threatening symptoms in cases of operation in which healthy recent omentum has been ligatured is not very difficult to explain. You will see at once in these cases that there is for the time being a state of affairs resembling strangulated omental hernia, a portion of omentum having been strangulated by the ligature applied around it ; thus the symptoms resulting from acute omental strangulation may arise. These however soon pass off because, unlike the condition which holds good in an omental hernia, the strangulated portion is not fixed by its implication in the hernial sac. Such is the reasonable explanation of the cases in which the recurrent symptoms come on shortly after

Digitized by Google

operations in which healthy omentum has been ligatured. But what is the explanation of the symptoms when they come on some time afterwards, intervals of as much as a fortnight perhaps intervening? The first man, for example, of whom I spoke remained well for a fortnight before these threatening symptoms supervened. It is not so easy to explain these cases, and I am not sure that we know the true explanation of them. One thing, however, is quite clear—that in cases in which ligatures are applied to old altered insensitive and hypertrophied omentum, what is called fat necrosis very commonly occurs in the stump which has been ligatured. Now if this fat necrosis is rather gradual in onset and of the molecular sort, it will give rise to very little trouble at all; but if the process is more rapid, and if in persons whose powers of resistance against micro-organisms are not very great, local inflammation later on happens, giving rise to adhesions or other sources of irritation, it is easily conceivable that symptoms of the remoter kind may follow. It is also probable that if the formation of adhesions explained the symptoms, that these would arise upon the consolidation of the adhesions, which would, in the ordinary course of events, be at the end of a fortnight or thereabouts from the time of operation. As soon, however, as the gut becomes quite untrammelled by inflammatory conditions, and begins to move freely, the adhesions are by degrees either broken or stretched, and the symptoms arising from them pass off. Whether that be the true explanation in all cases I cannot say, because I have not seen the inside of the abdomen in any cases of this kind; but in one case, in which death followed in a case of this sort from cerebral hæmorrhage three weeks after the occurrence of symptoms of the kind mentioned, it was quite clear that recent adhesions had existed, and had become stretched and broken through. A very important point about these remote cases is the absence of any rise of temperature. There are vomiting, pain, and perhaps distension, but the temperature does not go up; indeed the condition is exactly like that which is found in intestinal obstruction in the earlier stage. If there be a rise of temperature to an extreme degree in one of these remote cases, the outlook is not so reassuring, but in cases where the omentum only has been dealt with, it is well to be very chary about making a second exploratory operation. I have more than once known the abdomen opened in cases like these with an entirely negative result so far as objective conditions were concerned. In one case the patient died, in another recovery followed, and in the latter the operator received the credit of the recovery; my impression is that if neither had been operated upon both would have recovered.

Although I said just now that in the majority of these cases in which symptoms of this sort arise, the case may be regarded with complacency, that is not always so. I do not wish to leave you with the idea that in these omental cases no harm can ever arise, because harm does sometimes come of them, and the following may be the way of it. You will remember I said just now in dealing with the first case, that I was very careful not to apply the ligature to the fresh or recent omentum, because there was so little of it that the ligature would have to be applied very closely to the gut. In all cases in which you have to deal with omentum, no matter whether the omentum be altered or not, you must leave a sufficient interval between the ligature and the bowel; never if you can possibly avoid doing so, in cases like that I have mentioned, tie a piece of omentum very close to the bowel; it is a dangerous practice, and harm may come of it. If you cannot get one inch or at least half an inch away from the bowel, place the ligature around altered omentum rather than round healthy fresh omentum.

Not long ago, in private practice, I saw a case which was a very unfortunate one; and I have so often told you that you cannot judge of the value of treatments or procedures by simply mentioning successful cases. I therefore take this opportunity to relate what I call an unsuccessful case; indeed, it amounts to more than that, for it is an example of a surgical disaster. A man twenty-six years of age had a hernia, and he came to me for the performance of the radical care. The hernia was omental, and not remarkably large. The omentum in the sac was very hard—so hard as to be almost cretaceous,—and any ligature, however carefully applied, cut its way directly through, free bleeding following from the divided vessels. In this particular case, therefore, although I knew it was not under ordinary circumstances a good practice, I was really compelled to apply a ligature around the omentum less than half an inch from the gut.

Digitized by Google

The patient did very well for two or three days, and then he became very ill. He vomited; his temperature first became subnormal and then rose to 104°; his tongue became black, his lips dry, and he was evidently dying. Before his death it was quite clear what had happened; one could diagnose the condition with certainty as gangrene of the bowel; but the patient's state precluded the possibility of useful radical interference. The gangrene undoubtedly in this case arose from the nearness of my ligature to the gut. I do not know that this could have been avoided, but that it was the cause of the death there is no doubt whatever, as was afterwards proved by *post-mortem* examination. The case is an important one from a very practical point of view, since it shows that it is sometimes impossible, however careful you may be, to avoid disaster. If I had to deal with that case again I should probably act in exactly the same way; but it does not follow as a matter of course that if I did there would be the same termination. The case is especially important for mention here because it shows that, although as a rule the symptoms following interference with the omentum may be regarded with complacency, you may occasionally find a case in which they are of grave import. You will always have indications of the gravity of the symptoms: a high temperature, that is to say a temperature of 104° or so, with a very dry tongue; and especially should the symptoms be regarded as ominous if the high temperature be preceded by a subnormal one,—quite a different state of affairs you will note to that which I have described in the case of the man in the Hunter Ward.

With regard to the management of the omentum in these cases in which its removal is necessary, there is a very important detail which it is well that you should bear in mind:—the ligature used in tying omentum should always be stout, whatever the nature of the ligature may be, whether it be silk, catgut, or kangaroo tendon,—it can hardly be too thick. The reason for insisting on this point is the following:—you must understand that omentum, even when healthy, and especially when hypertrophied, is easily cut through by a ligature. Altered and hypertrophied omentum is sometimes so brittle that a thin piece of silk will cut through it as it would through cheese. This may not at first sight seem to be of very much importance, but it is really of the greatest moment. In hypertrophied or altered omentum, such as may be found in many hernial sacs, the walls of the vessels are very thin; the omentum itself having become more or less stiff, some of these vessels become canalised, resembling certain vessels in the liver. You will therefore readily see that if a piece of thin silk or other substance is tied tightly around a piece of tissue of this sort it may very quickly cut through it, together with the thin-walled gaping vessels in it, with the result that if the mass be returned into the belly before this accident is realised by the operator the patient may bleed to death. This is not an imaginary danger. I remember being present at an operation in which a piece of omentum was tied in the way I have said. The operation was completed, and there seemed to be nothing remarkable about it. But before the surgeons left the house they were called back to the case because the patient was in an apparently desperate condition. He was collapsed and pale, had a failing pulse, and in point of fact was presenting all the symptoms of profuse internal hæmorrhage. Under these circumstances there was only one thing to do, namely, to re-open the abdomen. It was found that the ligature, which had been applied to a piece of hard omentum, had cut through the omentum, and had also divided a large vein, and from this large vein blood had poured out so quickly that the lower portion of the belly seemed almost full of blood. The torn vessel was picked up, a new ligature applied, and the parts brought together again. Although the patient recovered, the accident was a very grave disaster, due to the fact that the ligature used was so thin that it cut through the brittle omentum. These observations as to the desirability for the use of thick ligatures are not confined in their application to omentum, but apply with equal force to the ligature of piles, etc. A stout ligature will secure the parts as well as a thin one, and there is no danger of its cutting through.

The point, however, which I especially wish to impress upon you now is that there is a liability in every case of omental hernia in which the omentum is ligatured, to the occurrence of certain symptoms subsequently, which although at first sight of a threatening kind, are really of little importance if the right treatment is adopted.

As a natural sequence, we now come to a consideration of the treatment of symptoms which are sufficiently indicated in the cases I have described.

Digitized by Google

The important factor in the cases is abdominal distension. Whether the symptoms come on immediately after operation, or whether they arise at the end of ten days or a fortnight, the first thing to be dealt with is the distension of the belly, and upon your success in the management of this condition the ultimate issue of the case mainly depends. When vomiting is co-existent—as is generally the case,—a large enema containing turpentine, or, better still, the enema rutæ of the St. George's Hospital Pharmacopœia is generally effectual. I strongly advise you not to rely upon the long rectal tube; you will rarely find it of much use. If the patient is not vomiting, by far the best plan is to give a large dose of castor oil with fifteen drops of laudanum; this is, so far as I have seen, better than the frequently repeated doses of salines (*e. g.* sulphate of soda) at frequent intervals, which is in favour with many people. The crucial point in the treatment of all abdominal conditions after operation is to avoid intestinal distension; and if in the course of your practice you take care to do that, you will find that very little trouble will arise in your abdominal cases, unless, of course, there is some very grievous condition which is incurable. On the other hand, if you ignore the oncoming of abdominal distension, no matter how well the patient may seem to be otherwise, trouble will undoubtedly arise. Symptoms will, under such circumstances, sooner or later occur which will give cause for great anxiety, even in cases which seem to be otherwise doing well. If care is taken to secure a free evacuation of wind or fæces soon after an abdominal operation, you will always save yourself and the patient a great deal of trouble, and sometimes avoid a serious complication or disaster. Nothing is so bad in abdominal work as to be afraid of allowing the bowels to act soon after the operation.

A symptom which attracts much more attention at the hands of many practitioners is vomiting; but vomiting in itself is of comparatively small importance in the absence of distension; vomiting without distension is of little moment; vomiting with distension is of momentous gravity. The indication in such circumstances is the treatment of the distension and not of the vomiting, as is so commonly adopted; if the distension can be relieved the vomiting instantly ceases, and no treatment applied with a view to the arrest of the vomiting will save the patient if distension is unrelieved. Of course I am not now speaking of vomiting following the administration of an anæsthetic, which is outside my present subject, since it is unaccompanied by abdominal distension.

Bear in mind that whatever the cause of abdominal distension may be, that if the distension progresses beyond a certain point, paresis of the gut follows, which will lead to meteorism, and if meteorism occurs in these cases, the danger to life at once becomes an objective factor, which at times requires no ordinary skill in its management.

You will recollect that in the case which I described of the man in Hunter Ward, when we found the threatening symptoms occur, he was given enemata as freely as possible, as well as medicine by the mouth, in order to get a free intestinal evacuation. Had we been misguided enough to have given that man opium or morphia in order to allay the pain, and to have ignored the condition of his intestines, the distension would have increased, and he would almost certainly have died, especially as he suffered, as many of these patients do, from chronic bronchitis with emphysema. With such pulmonary complication it is more than ever necessary that every little increase in the abdominal distension should be carefully noted, and attempts made to diminish it, because additional distension of the abdominal cavity, to however small a degree, results in diminution of the breathing capacity, a frequent complication in these cases, and one of very great moment, since, if its gravity is not realised, a fatal termination may follow almost before the practitioner has realised the onset of serious symptoms in the case. Time prevents my dwelling further at present upon a subject which is of much surgical importance, but I trust that the lessons which I have endeavoured to impress upon you may not be altogether valueless.

LACHER reports the case of a man who, as a result of chronic empyema, developed marked swelling of the end phalanges of the fingers and toes, involving exclusively the soft parts. An operation reduced the size of the abscess cavity, and as a result, the breadth of one thumb diminished from 3·2 cm. to 2·7 cm. and the other fingers in proportion.—*Philadelphia Med. Journ.*, July 6.

Digitized by Google

TWO CLINICAL LECTURES ON INTESTINAL ULCERATION.

Delivered at the West London Hospital.
By C. R. B. KEETLEY, F.R.C.S.,
Senior Surgeon to the Hospital.

LECTURE I.

GENTLEMEN,—I am afraid I shall find it difficult to make what I am going to say interesting to you, though it is very interesting to me. The cases are of very great importance, especially in reference to general practice.

I have had sent on to me this afternoon a letter which illustrates this. It is about a personal friend of mine, who is himself a medical man. He lives some hundreds of miles from London, and a few months ago he was suddenly taken with intestinal obstruction. He had been noticed for a long time to be looking unwell. The surgeon who was called in was a well-known man in the district, as well as in London. My friend is very stout, and as the surgeon did not know where the obstruction was, and the case was dangerously acute, he did a colotomy in the right inguinal region; and I think he opened the cæcum. That relieved the obstruction and, luckily, saved my friend's life. He must have been very bad, for he had hiccough, which is a serious sign in cases of intestinal obstruction. He is relieved but not getting well; and as he has all along rather wished to see me, this letter which I will read to you was sent to a mutual friend, and came to me to-day. He says "he has not been progressing so well as we should have liked; the fæces have been very liquid, and have caused a good deal of irritation." You will remember I said he had colotomy done on the right side, and fæces have a tendency to be rather softer there than in the ordinary place, because it is very close to the small intestine. "But the irritation is better." I should say this letter is from the family doctor. "He is going to have a new colotomy pad fitted on to-night, and he hopes to go to the seaside on Wednesday for a few weeks. The surgeon who operated was over last Tuesday. He is inclined to think that the cause is malignant, but he will not give a definite opinion. Of course the abdomen is very difficult to palpate. There is still some tenderness in the sigmoid region—he was not operated on in the sigmoid region,—and although there is nothing distinct to be felt, there has been some fluid in the abdominal cavity. He has had several enemas, but nothing of any consequence has come away. On Saturday he passed a little blood and slime *per rectum*, without any straining, and, unluckily, he saw it. He now seems to think he has some malignant trouble, but he has not the appearance of a person suffering from malignant disease, although the history points that way. He wants to go up to London." I am sorry to say I have scarcely any doubt but that this is a case of malignant disease of the large intestine, and very likely it is the sigmoid flexure or the descending colon. Looking at such a case in the abstract, it ought to be operated on as soon as possible, with a view to finding out what is the matter. Probably the reasons why nothing of that kind has been done were, in the first place, that he is a medical man himself, and therefore not very keen about being operated upon; secondly, he is a very stout man, so nothing definite has been felt; and thirdly, the diagnosis is only guesswork, although I believe the diagnosis is so likely as to almost amount to a certainty. Those reasons, if I may call them so, constantly lead to such cases being treated in a palliative way until they have got so far that it is of no use doing anything to them after all. I know of a case in another friend of mine, who was a member of the medical society which meets here. He had such a history as this, and he consulted eminent surgeons, and, for I suppose similar reasons, they did not come to any definite conclusion. At last, when it was perfectly plain what was the matter with him, the disease had advanced in such a way, and so much adhesion had occurred, that it was ridiculous to hope to do any good by operation. He had no radical operation at all. This gentleman, if only sufficient determination had been shown in dealing with his case at first, might have had his life saved, because, curiously enough, these malignant ulcerations of the large intestine do not seem to have a very early tendency to infect the system, and even when they are of considerable size, sometimes the glands are found to be free. One thing especially which leads to patients being treated in a palliative way, owing to uncertainty of the diagnosis, is that the patients are often very stout and often they do not lose weight for a long time. Now, early this year, a man came to the

Digitized by Google

hospital who, but for the expression of his face, was very healthy and very stout. He was a publican, but not an alcoholic. He came from a town in Lincolnshire, and his case was first diagnosed there by Dr. Wood. He obviously had some obstruction, some ulceration in his large intestine, because he passed blood and mucus in his stool, and nothing could be felt by the rectum. But there was a distinct feeling of something in the left iliac region of the abdomen. I opened the abdomen and found he had a very considerable tumour of the sigmoid flexure, and there was no difficulty at all about taking it away. Ever since then he has been practically well, that is to say, ever since the completion of the operation, because I did it in three stages. It is quite likely that he may never be affected again.

I have commenced my lecture in this way to show that the subject is a very interesting one, though I did not intend to plunge straight into the consideration of malignant disease of the intestine.

Forms of intestinal ulceration.—You know that there are a great many forms of ulceration of the intestine. The intestine, like any other part, is liable to be ulcerated by traumatic influences. For instance, a person may have what is called an intestinal calculus. These calculi sometimes grow to a considerable size, and they are apt to lodge in some particular place, where they may produce ulceration. Of course they are not very common. Another form of ulceration in the intestine, which is more frequent, occurs when a gall-stone ulcerates into the intestine. Intestinal calculi often have a gall-stone as the nucleus. Then foreign bodies passed in through the mouth lead to ulceration, and it is astonishing what curious and large bodies will find their way out of the system without doing any harm. I remember one evening as I was going to leave the hospital being taken to a servant girl who had swallowed a hat-pin with an unusually large knob. She had swallowed it a fortnight before, but when she said so she had not been believed. When I saw her the pin was sticking out of the left iliac region, and an attempt had been made to pull it out, but the knob anchored it. I made an incision to enable my finger to go to the end of it. I supposed this end to be in the intestine—I thought in the descending colon—and I hoped to be able to pull the pin out of its knob, letting the latter fall back into the intestine. I worked with that intention, but I was not successful; I was therefore obliged to pull the knob and pin out together. The girl got well without any trouble. I do not know whether the pin had pierced the intestine or the stomach,—very likely the latter.

The rectum is the place where foreign bodies most frequently cause ulceration. For instance, fish bones may be felt at the bottom of an ulcer in the rectum, and can be taken out without any difficulty. I remember in one of these cases I used cocaine, soon after it first came into use. We only used a grain and a quarter of cocaine altogether. We injected half the solution into the submucous tissues, and used the other to soak cotton wool, and place in the rectum. It gave the man alarming temporary cocaine poisoning; he seemed to have very great difficulty in respiration for some minutes, and appeared to be breathing almost entirely by his voluntary muscles. But it passed off, and we (*i. e.* Mr. R. J. Lloyd and I) did not have any further trouble. I do not know whether the rectum is especially subject to poisoning from cocaine. One of the most famous cases of cocaine poisoning known is that in which a celebrated St. Petersburg surgeon used it and the patient died, and the surgeon then committed suicide.

I remember another instance of traumatic ulceration of the rectum. It was that of a boy who fell out of a window, or else off a roof; at all events he fell into a thorn bush, and a piece of thorn as thick as a lead pencil, and four to five inches long, went through his anus and into the rectum, causing an abscess, which abscess discharged both into the rectum and into the buttock. I got the thorn away, and the patient quickly recovered.

An ulcer of the intestine sometimes forms by the discharge of an abscess, which has nothing to do with the intestine to begin with. We all know that many abscesses connected with the female genital organs discharge into the large intestine, some of them into the rectum. Some open into the small intestine. I had a case here for a long time which opened into both small and large intestine, and a very troublesome case it was, requiring enterectomy. Of course in all the classes of cases I am now speaking of the treatment is essentially the removal of the cause. When an ulceration of the intestine forms in

Digitized by Google

consequence of abscess pointing into it, it is not of much use refreshing the hole in the intestine, and sewing it up. The thing to do is to cure the fistula, scrape it out, and follow out every branch, then entirely clean and disinfect it, and provide a free opening to the outside of the body. Then the hole in the intestine has a natural tendency to close up. Of course some cases in which an abscess opens into the intestine are very serious ones, and one often comes across them too late to do them any good. I remember a woman who came into the hospital some years ago. She was a very fat woman, and the whole of her right side was infiltrated with pus. I made an incision, and found the whole of the external oblique, which could be exposed on the right side, was one great slough. Her urine was loaded with sugar, and she died in twenty-four hours. At the post-mortem we found her appendix vermiformis was sloughed, and that perhaps had been the starting-point of the whole mischief. She also had a large opening, big enough to admit the finger, in the ascending colon. That possibly was simply a case of ulceration into the colon from outside.

I mentioned ulceration caused by pressure of intestinal calculi. Hard pieces of fæces lodged in the intestine may cause ulceration in a similar manner.

Now, there is a class of cases which were very well described by Dr. Hale White some years ago, I think at the Clinical Society. Some American surgeons, and in this country Mr. Mayo Robson, have operated upon them and done them a great deal of good by opening the colon in the neighbourhood of the cæcum and washing out the whole colon from end to end. I refer to ulcerative colitis. The cases which Robson operated upon were cases of membranous colitis. But whether a membrane forms or not, there is a form of ulcerative colitis which is quite distinct from dysentery and from malignant disease and from various specific diseases. When ulcerations form in the colon they are very obstinate. The symptoms of almost all these ulcers are diarrhœa from time to time, and the passage of blood and mucus; also tenderness and pain in the abdomen. The degree of pain and tenderness varies very greatly in different cases.

Another common cause, really more common than those which I have mentioned, of ulceration of the intestine is tuberculosis. Tuberculous ulcers form in any part of the intestine, but perhaps more frequently in the upper part of the rectum, and in the cæcum and ascending colon. Sometimes they form in the appendix itself. A case in point was that of a boy, from whom I removed the appendix yesterday. He had an ulcer, probably tuberculous —the histological examination has not been made yet—at the distal extremity of his appendix, an ulcer the size of a shilling, and it caused him to have attacks of pain and appendicitis. His case was first diagnosed by Dr. Gilbert Richardson. One of the commonest seats of ulcers caused by foreign bodies is the appendix.

The way to get tuberculous ulcers better is to rest the intestine by opening it above the ulcer, and then to scrape the ulcer itself, and try by that means to get it into a healthy, clean state. But in many of these ulcerations we do not get an opportunity of doing this, though tuberculous ulcers are generally low down in the intestinal tract. A young woman was in here a few years ago with a very extensive tuberculous ulceration just about the junction of the sigmoid flexure with the rectum. It began as high as one's finger could reach. Attention was called to it, not by anything which came from the rectum at first, but by the fact that fistulæ, which led from it, formed an abscess, which burrowed and opened everywhere about the buttocks and the upper part of the thighs externally. In fact, she had a large number of fistulæ. All these fistulæ and the ulcer itself were scraped. The abscess entirely surrounded the rectum, and went up as far as I could expose it by means of a long speculum, after a posterior linear proctotomy. An inguinal colotomy was performed. When she came here she had had this disease for a long time, and a hectic temperature, but gradually she got perfectly well. I often see her about now riding a bicycle. She has remained well for several years.

I may now say a few words about some things which do not concern the surgeon directly, but which one ought to mention because they result in ulceration. There is the ulceration which proceeds from dysentery. When that calls for surgical interference it is generally by way of the liver. I need not remind you that it is a common cause of abscess of the liver. But it also sometimes causes stricture of the intestine, which is not malignant; and I cannot help thinking that it also

Digitized by Google

sometimes leads to strictures which are malignant. Perhaps any common place cause of ulceration of the intestine, with the exception of tuberculous ulceration, may ultimately lead to malignant disease. Then syphilis is another cause of intestinal ulceration. Syphilis affects especially the lower end of the large intestine, that is to say the rectum, but it does not entirely confine itself to that part. Some years ago I had an elderly gentleman under my care. He had a distinct tumour on the left side of his abdomen in the lower part, a little higher than the left inguinal canal. I opened the abdomen and found the tumour was a large one, the size of a hen's egg, though not nearly so definite in shape. It was closely adherent to the omentum, to the small intestine, to the sigmoid flexure, and to the bladder. It was especially its association with the bladder that deterred me from removing it. I would not have minded taking a small piece of the sigmoid flexure away, but I hesitated to make a large hole in his bladder, especially as I thought the disease was malignant and that it had gone so far that it would certainly recur. He had a history of syphilis, and I hoped his trouble might be syphilitic. Large doses of iodide of potassium, twenty grains three times a day, completely cured him. He retired, and went to live at the seaside. One day he sent for me in a great state of alarm, as he was convinced he had malignant disease of the bladder. He had not got that, but an enlarged prostate and its results, and he eventually died of some form of general mental and nervous degeneration.

Another possible cause of intestinal ulceration is worms. It is perhaps a little uncertain as to whether the worms cause the ulceration, but at all events worms are a not infrequent complication of ulceration, especially in the neighbourhood of the cæcum, and in those cases in which an immense ball of worms has been found, so large as to cause symptoms of intestinal obstruction, there can be no doubt that the ulceration has been caused by the worms. The most common kind of worm which has been found escaping from ulcer of the intestine is the round-worm. Our late house surgeon, Mr. Maitland Thompson, published such a case, on which he had operated successfully.

I said just now that the signs of intestinal ulceration are pretty much the same, whatever may be the cause of it. Of course in some special instances, as in the case of typhoid fever, there are quite special symptoms independent of the ulceration, which are superadded. There is the fever of typhoid, and in tropical countries in chronic dysentery amœbæ coli are to be found by microscopic examination of recent fæces. As I have said, the ordinary symptoms are diarrhœa and then there is a limited quantity of pus, and occasionally mucus, occasionally blood, and even shreds of tissue found in the evacuations. The fact of the quantity of pus being limited is a point of great importance. If there is a large quantity of pus you may be almost sure it does not come from an ulceration of the intestine; it must have found its way from the outside, from an abscess or fistula. Then you have to take into consideration the whole history of the case, and all the symptoms which you can discover by careful search. If the patient be a woman, and she is comparatively young or not too advanced in age, you will inquire into the state of the genital organs and the history of these parts. If the patient be a girl, or a boy, or a young man, there is likelihood of it being tuberculous in origin, and, of course, here the microscope might assist very greatly. You might also find disease of bone in some region, especially in the pelvis. Do not omit careful rectal or vaginal examination. The pain varies very greatly. There are some chronic ulcerations of the intestine which are almost free from pain. Tuberculous ulcerations especially are often anything but painful. On the other hand, some are very painful.

Perforation.—Of course one of the worst things that can happen to complicate ulceration of the intestine is perforation. I do not propose to talk about appendicitis to-day, although a great deal of what I am going to say about ulceration applies also to perforation of an ulcer in the appendix. But putting that aside, perhaps one of the commonest causes of perforation is a typhoid ulcer. I am sorry to say that my experience of that is not very great. I suppose in this hospital the physicians cure the cases of typhoid before perforation occurs. I was called a long time ago to see the wife of one of the gentlemen who was on our board of management. She had had perforation following typhoid, and the perforation had existed for some days. I opened her abdomen, and almost the first cut exposed the perforation; the perfora-

Digitized by Google

tion almost presented in the wound. It was a small ragged hole, one third of an inch in diameter, and I cut the ulcer out and sewed the part up. But the whole of her peritoneum was in a state of suppurative inflammation, and the poor lady died.

Another case was that of a girl about eleven years of age, who ten years ago was brought in here with a diagnosis of typhoid fever, and the temperature chart and the history justified that diagnosis. She had the whole of the lower half of her abdomen full of pus, and adhesions had cut it off from the upper part. I opened the peritoneal cavity, removed the pus, cleaned it out carefully, and put in three iodoform gauze syphon-drains. One long drain I passed into the right flank, one into the left, and one into the pelvis, together with a tube. The patient ultimately recovered, thanks in a great measure to the care which was taken with the case by my then house-surgeon, Mr. Sidney Bontor. Her symptoms very suspiciously resembled typhoid for two or three weeks; she had a great deal of delirium, the temperature went up in the evenings, and always came down in the mornings; but the diagnosis could not be absolutely verified as, fortunately, there was no opportunity for a post-mortem. The patient enjoyed robust health ever afterwards.

Ulcerations of the intestine may perforate into other cavities, such as the bladder. A few years ago I had occasion to operate on a retired naval captain. He was a very sensible man, at which remark you may be surprised when I tell you the history. He had a tumour which could be felt in the sigmoid flexure. His rectum was full of small polypoid growths. I might mention that multiple polypoid growths in the rectum are not very common; perhaps not more than a dozen cases have been reported, but they are apparently apt to be complicated by ulceration, probably of malignant nature. Well, I cut down upon the abdominal tumour, and found it was a tumour of the sigmoid flexure. I opened the sigmoid flexure, and found it was a circular ulcer which went almost round the flexure. And even as high as this ulcer, by passing my finger through the ulcer, I felt one of these polypoid growths close to it. I found he had a number of glands enlarged, and that led me to rather hesitate to do a radical operation upon him. I ought to add that this tumour was adherent to the bladder, which of course complicated matters a good deal, and I could not be sure it was not adherent to other parts, such as the left ureter. Therefore I contented myself with doing a sigmoid colotomy. This was done in the country, and I had hoped to see him again in town with a view to reconsidering the question of doing a radical operation. But he came under certain influences, and was persuaded to go to a notorious quack, a blacksmith by trade, to have injected into him some irritating fluid. The quack commenced to inject this stuff into the colotomy wound, and continued to do so. He also injected it into the rectum, and quantities of mucus, blood, and possibly shreds of mucous membrane and pieces of tumour came away. The poor patient accordingly thought the whole process was curing him, and that the disease was coming away. He was told that this was so, and so he persevered with the treatment. Suddenly one day perforation took place into the bladder, and fæces began to come by the urethra. That did not destroy his confidence in the quack, and so the quack commenced to inject his nostrum into the bladder. He was not able to pass a catheter himself, but the nurse passed the catheter for him, and the injection caused the unfortunate patient excruciating pain and agony. At length he could not stand it any longer, gave up the quack, and sent for me. I now found that the growth had extended enormously. It was much larger than anything of the kind I had ever seen before. It occupied two thirds of the left side of the abdomen, extended over to the right side, and was practically blocking up the colotomy wound, so that I was obliged to do another colotomy. I did what occurred to me as a good way of performing colotomy in such circumstances. I simply pulled out the part of the transverse colon near its hepatic flexure near the right groin, and treated it exactly in the same way as a sigmoid colotomy. So far as the intestinal obstruction was concerned, the patient was relieved.

I may point out that with a mesocolon of average length, the transverse colon near its hepatic end may be brought out of a wound nearly as low as the right groin, and a good spur secured even there—a desideratum not easily obtained with a cæcal colotomy.

Digitized by Google

SOME REMARKS*

ON

THE AFTER-EFFECTS OF ANÆSTHETICS.

By J. BLUMFELD, M.D.Cantab.,

Assistant Anæsthetist, St. George's Hospital; Anæsthetist to the Grosvenor Hospital for Women and Children.

NOT much attention is generally paid to the question of what results may be produced by the taking of an anæsthetic beyond that of the anæsthesia for which the agent is administered. Yet apart from the physiological interest of the matter it is also of some practical importance, particularly for the general practitioner, in whose hands mainly lies the care of patients after operation. In hospitals, however, an anæsthetist has ample opportunity for studying the after-effects of his administrations, and it is mostly from such observations, together with inquiry and experience in private cases, that these few remarks are offered.

After-effects, generally only of trifling degree, are at present, in many cases, a necessary evil, subsequent upon the taking of any of the general anæsthetics that we can use for maintaining long anæsthesia. We shall mention the different effects that are produced after the common anæsthetics, and touch upon the methods by which they may be, as far as possible, prevented or reduced to a minimum.

Sometimes the fear of sickness afterwards plays an important part in a patient's dread of taking an anæsthetic, and in this way, too, it becomes of importance to be able to assert that this or any other unpleasant consequence will be of the shortest possible duration, if present at all.

Some general considerations of preparation before, and treatment after, the administration of anæsthetics apply in all cases, and may be dealt with before discussing different agents severally. The ordinary rules for preparing a healthy patient for operation by securing an action of the bowels the day before, giving no food for six hours previous to operation, etc., are familiar to you all. In the case of infants the preliminary starvation is often too rigorous. An infant at the breast should be fed not more than three hours before operation. I have seen serious collapse after circumcision that was due only, I believe, to the fact that the infant had not been fed for seven hours beforehand. Similarly, after operation, unless some special reason against it holds good, the infant may advantageously be put to the breast at the end of an hour after consciousness returns. Artificially-fed infants should generally wait two hours. It is important that every part of the patient not necessarily exposed for the operation should be carefully covered with a blanket, or warmly clad. This precaution has an important bearing upon subsequent shock or bronchitis. In cases of intestinal obstruction, where there has been vomiting, the stomach should be washed out just before the anæsthetic is administered. Very large quantities of dark gritty fluid will sometimes be removed in these cases, which would otherwise have been quietly regurgitating into the patient's mouth and nose throughout the operation, and would possibly give rise to trouble afterwards, through being inhaled into the air-passages in small quantities. This material probably comes largely from the intestine, not from the stomach, for it is alkaline in reaction, and consists mainly of, besides water, hæmatin, ferments, and altered blood-pigments. In the case of patients who are collapsed, if ether is to be given, no special preliminary step is necessary, the stimulation of the anæsthetic being enough. If chloroform is to be used, a hypodermic injection of ten drops of liquor strychninæ should be given before the anæsthetic, and when the patient is under, one or two pints of water at 105° with two ounces of brandy may well be slowly injected into the rectum if thought necessary.

As regards treatment immediately after operation, the chief points to be considered are—the moving of the patient, his feeding, and the atmosphere of the room. Whenever it suits the surgeon equally well, the operation should be performed upon the bed in which the patient is to lie. In this way there is no need to move him about, which is a great gain, and increases the chance of escaping all unpleasant after-effects. Many minor operations and nearly all rectal operations can be thus performed. In the latter cases the patient is on one side, with the buttocks at the edge of the bed. When the surgeon has finished, the patient is simply lifted a little further on to the bed, remaining on his side, with a pillow at his back.

* Delivered before the Chelsea Clinical Society.

Digitized by Google

This is the best position for all cases after operation, when there is no particular reason against it from the nature of what has been done. When the patient has to be removed from the table to the bed, the head of the table should be placed at right angles to the foot of the bed. Those lifting the patient stand between the bed and the table, put their hands under him, turn round and deposit him slowly, taking particular care not to let the head flop about. Shaking a patient by carelessly moving him certainly increases the chance of after-sickness. The room should be kept at a uniform temperature, without draughts, with a window open at the top, quiet, and with the blinds down. In all ordinary cases the patient should have nothing by the mouth for six hours, except sips of hot water. The first food should be beef-tea or thin soup, or tea and some dry toast. When there is much sickness, hot black coffee is useful, and inhaling fumes of vinegar or strong sal volatile. Sometimes strong brandy and soda or champagne answer better. In exceptional cases a hypodermic of morphia stops otherwise uncontrollable vomiting. With many men milk is the favourite thing to give as the first food by the mouth after operation. It certainly should not be, for milk is more likely than many other liquids to provoke vomiting in a person who has recently taken an anæsthetic. This applies particularly to persons who have taken ether, for there is then almost always after the operation a certain amount of mucus in the stomach that was swallowed when the patient was unconscious. This mucus is more easily vomited or passed on into the intestine if diluted with hot water; if milk is added to it, there tends to be formed a stringy, cheesy mass, which almost certainly provokes laborious vomiting.

We shall now briefly discuss after-effects systematically, as they arise in connection with the three commonly used anæsthetics, viz. gas, with or without air or oxygen, ether, and chloroform.

A few general remarks apply to all cases after an anæsthetic. *Firstly*, the nature of the individual has a great influence in determining the nature of the after-effects. It often occurs to an anæsthetist that he is asked to anæsthetise someone who has already undergone an operation, and has suffered much afterwards—let us say from vomiting—and that ether was the agent used. The patient is anxious not to undergo again the nausea that he enjoyed so little and experienced so long on the first occasion; he accordingly requests you to use chloroform, and as you find nothing in his condition to contradict it, you agree to do so. You will now certainly be wise if you tell him that you cannot promise that he will be more free from sickness this time than he was last. The probability is that, granted, of course, that he has to be kept under as long as before, his sickness will be just as severe. There are some subjects in whom at the close of the operation one can say almost certainly, he will not suffer at all from after-effects. Such are the big healthy men subject to much outdoor exercise, large amounts of alcohol, and good living generally, who take a great deal of the anæsthetic, and are by no means the most convenient subjects to anæsthetise. I have seen a gentleman of this kind, after taking ether and being surgically under its influence for nearly half an hour, sit up within three minutes of the close of the operation, talk perfectly rationally, and feel not the slightest nausea. He had no after-symptoms at all, except a certain amount of headache. Even this, I believe, need not have been experienced, had he been willing to rest quietly, and take a less active interest in an account of the operation and the cleaning up of the surgeon's instruments.

Again, the rule applies to all anæsthetics that, generally speaking, the more of the agent used, the more likely is the patient to suffer afterwards. Herein, of course, lies the great secret of prevention of after-effects. Compatible with perfect anæsthesia, use as little of your agent as possible, and it is astounding in this respect what a great difference practice makes in the possibility of conducting a long administration with comparatively little anæsthetic.

Thirdly, the preparation of the patient is with all anæsthetics a matter of much importance.

Roughly speaking, the common thing is after gas no after-effects, beyond an obscure sense of uncertainty as to when consciousness was lost, and of dreaming; more rarely there is headache or some giddiness or faintness and slight nausea, lasting occasionally for quite a considerable time. In such cases there is pallor and much sweating. I have notes of one patient, a medical man, who remembered distinctly that he experienced great pain in his head just before he lost consciousness. In him

Digitized by Google

severe headache lasted for several hours. Very occasionally there is vomiting even after a short gas administration, and this is less uncommon after gas and oxygen; there is also more often excitement of a somewhat hysterical nature after the use of this mixture. There may even be vomiting during the administration—and this even although the last meal was several hours before. Very occasionally after gas the return of consciousness is long delayed. In one such case a young woman lay for over an hour quite unconscious. The pulse, colour, and respiration were natural, and she was obviously in no danger, but simply could not be roused; later she recovered perfectly and with no further ill-effects. Curious emotional states are sometimes witnessed after gas, though just as with other after-effects, they are more common after ether and chloroform. There may be weeping for some time or, more happily, I have seen laughter of an extremely hearty character.

With ether or chloroform, the after-effects might be considered according to the part of the body that is affected. Most usually there is evidence of after-effects upon the *central nervous system*, particularly its highest portions. There may also be visceral effects, and effects upon the circulatory system. The most common symptom, next to the evidences of partially restored consciousness, such as purposeless movements and incoherent talking, is certainly retching and vomiting. Very often, consciousness returns with the first attack of vomiting. Afterwards there is during the next few hours, some retching and nausea. Perhaps some more mucus and bile-stained gastric juice is vomited. The ordinary vomiting is less common after chloroform than after ether, but the cases of very severe vomiting, difficult to control, which occasionally occur, are generally due to chloroform, not to ether. Vomiting is probably a central effect and, not as some hold, largely a local affair due to the mucus swallowed during operation. This has a little influence certainly, and one is consequently able to do some good by lessening as far as possible the swallowing of this mucus, and by removing it with a stomach-tube; but even with these measures and careful preparation of the patient one cannot secure the absence of vomiting. Moreover, when ether is administered by the rectum vomiting still occurs; for these reasons I think it should certainly be regarded as a result of the influence of the anæsthetic upon portions of the brain just as much as is the disordered mental condition of a patient emerging from anæsthesia. Very commonly vomiting takes place entirely before the return of consciousness, and the patient is not aware that he has been sick at all. A more grave, and happily, a much rarer central effect is insanity. Anæsthetics alone may produce insanity, or it may depend upon the anæsthetic and the operation. One anæsthetic is not, according to Dr. Savage, more likely than another to produce insanity, and he has seen mania follow the administration of gas for a dental operation. Stupor lasting for weeks has occurred in several cases and, according to the same authority, there is most risk of this after anæsthetics for operation upon bladder or rectum. Anæsthetics are drugs which produce temporary mental disorder, and it is only natural that occasionally disorder of a more lasting character should arise. Melancholia or delusional insanity are very rare; but mania may certainly occur, and I know of one such case in which it lasted for weeks, and have myself had one in which fortunately it was of only two days' duration. Anæsthetics to persons suffering from recurring attacks of insanity are very dangerous in this respect. As regards epileptics, I have never seen fits follow as an after-effect, though I have seen one occur during the administration—luckily, the operation, which was for wiring a fractured patella, had not yet begun.

Various paralyses have been described after anæsthetics. They may be of (1) peripheral origin, (2) central origin, (3) indeterminate origin. The first are merely mechanical effects produced by the position in which an unconscious person has been kept. Such are paralysis of the deltoid, the patient having been so placed that the nerves were compressed between clavicle and first rib. The second are really instances of effects upon the circulatory system, and are various forms of monoplegia and hemiplegia due to hæmorrhage in the brain, just as in ordinary "stroke." When we consider the congestion caused by ether it is astonishing that such accidents are not more common. Their possibility must always be borne in mind in choosing the best anæsthetic for a particular case.

Visceral effects are mainly those upon the kidneys and lungs. Both ether and chloroform may cause albuminuria—the latter more than the former,

Digitized by Google

yet persons with renal disease already existing are better subjects for chloroform than for ether. The liver is probably sometimes affected, jaundice having not infrequently followed anæsthetics, and the condition of biliousness is common. Chloroform may cause fatty or granular degeneration of muscle-cells throughout the body, of liver, kidney or ganglionic cells of heart, and of adipose tissue generally; such changes may have evil results long after administration. Ether is less frequently followed by cellular and visceral changes, as might be expected, since it has been shown experimentally by Waller to have about one seventh of the deadening effect of chloroform upon nerve-tissue.

Of the lung effects of ether much has been written, and the term "ether pneumonia" has been employed to represent them. This is a bad expression, for lobar pneunomia is a very rare sequel to the inhalation of ether, and in the few cases that have occurred, there has not only been no proof, but also no great probability that the pneumonia was due to the ether. When chest trouble follows the taking of ether, which happens to some extent —probably in about one case in a thousand—the form it takes is that of a coarse bronchitis, arising within the first two days after operation, and lasting a few days, during which the temperature reaches 101°. This sequel is most commonly seen in abdominal and breast cases, the difficulty of coughing up mucus, owing to the pain of the wound and to bandages, being no doubt partly responsible. The possibility of its occurrence should make us chary of giving long administrations of ether for these cases. From this point of view too it is highly important that patients should not be kept rigidly on the back, but should be propped up in bed or allowed to turn on to the side even a few hours after operation. There is a series of symptoms, sometimes arising within a day or two of operations, which has probably been responsible for some mistaken diagnoses of ether pneumonia. The symptoms are in all probability due to a small pulmonary embolism, as has been pointed out by Dr. H. Menzies. In the course of most operations, a certain number of ligatures are applied to veins, and it is not unlikely that now and then a portion of clot formed above the ligature may become separate, reach the right side of the heart and so the lungs. Here is a characteristic instance in which the symptoms which might readily have been mistaken for pneumonia were probably of embolic origin. In this case ether was only used to induce anæsthesia, along with gas, chloroform being substituted as soon as the patient was unconscious. It is practically impossible therefore that the patient should have had "ether pneumonia." A youth of seventeen was operated upon for appendicitis. Anæsthesia was induced by gas and ether, chloroform being substituted before operation was begun. There was no difficulty in the anæsthetisation, and nothing unusual in the operation, during which a chronically inflamed appendix was removed, several ligatures being applied to bleeding parts in the course of the operation. Recovery from the anæsthetic was good, and nothing unusual occurred till the morning of the third day after operation. Acute pain was then felt in the left side of the chest; respiration was quick and short, the temperature arose to 103·5°, there was sweating and there was a painful cough with expectoration of bloody mucus. On the following day there was well-defined tubular breathing near and above the level of the angle of the left scapula. The respiration on this day was already much easier and the temperature not so high. There was no further blood-stained expectoration, and after three days the temperature was normal. During these days the boy was quite comfortable, and no physical signs were detected except a few râles at each base. The after progress of the case was perfectly satisfactory. The suddenness of onset and disappearance of the pain, its short duration, the sweating, the entire absence of delirium, and the general appearance of the patient made it highly improbable that this was a case of pneumonia. There was probably a small pulmonary embolism due to a clot from some vessel ligatured at the operation, and such cases have probably been several times reckoned as cases of pneumonia after ether. Dr. H. Menzies has related similar cases after confinement when no anæsthetic had been used to which the "pneumonia" could be attributed. It has been thought possible to catch pneumonia from the face-piece or from the bag of the apparatus. Ether vapour is itself germicidal, so that the bag from which the patient breathes is a very unlikely source of infection, and a well-cleansed face-piece is less likely to harbour the pneumococcus than is

Digitized by Google

the patient's mouth, or the air of the room in which he lies. After operation, patients are, generally speaking, in a state of diminished resistance, and it is not surprising that they should occasionally fall victims to germs that are commonly present either in them or in the air they breathe. In this way, and through exposure, are probably to be explained the very rare cases of pneumonia following operation. These cannot be attributed to ether, which evidence shows to be guilty only of causing sometimes a certain amount of catarrh of trachea and bronchi; for this reason it is best not to use ether to any large extent in the case of infants and of abdominal operations. In all cases undue exposure of the patient at the operation, and afterwards exposure of him to low and fluctuating temperatures, and to draughts, must of course be carefully avoided. These causes help, if they are not mainly responsible, when bronchitis follows use of ether.

Another most important principle to be observed, and which has already been alluded to as bearing on all after effects, is to use as little ether as possible compatible with perfect anæsthesia.

In very rare instances, acute œdema of the lungs has been found in patients who died shortly after an administration of ether. Curious but unimportant effects sometimes following the inhalation of ether and chloroform are prolonged attacks of hiccough or of yawning. These are presumably due to disturbance of the respiratory centre, and various rhythmical variations in respiration may be observed when patients are closely watched throughout a long administration. In one case hiccough at very short intervals continued for fifteen days after the taking of an anæsthetic. The disturbance was cured by morphine. Epistaxis sometimes follows ether taking; it more usually begins before the anæsthetic has been removed, and soon ceases after its withdrawal; it is not of frequent occurrence.

Dionin in Disease of the Cornea.—Dr. L. Vernies uses dionin in all corneal diseases and those of the iris and ciliary body, where it is best applied in combination with mydriatics. He uses it in solution of 5 to 10 per cent. strength. Ulcers of the cornea heal rapidly under its influence. It acts especially by increasing the flow of the lymph. —*Jour. A. M. A.*, July 6, 1901.

TREATMENT OF FRACTURES.

Mr. John Poland has just published, through Messrs. Smith, Elder & Co., his Hunterian Oration, "A Retrospect of Surgery during the Past Century," delivered before the Hunterian Society this year. We extract the following passage from this scholarly production: "I may at once say that the methods of dealing with fractures are old and empirical; these injuries ought to be treated in a far more scientific manner than they are at times. It is not the methods which are at fault, but the principles actuating their employment. The treatment of fractures has been a matter of routine for very many years, and it is only lately that it has undergone any modification, as in so many departments of surgery, by the influence of asepsis and other modern advances. While recognising that early massage and ordinary methods of treatment, followed by an early recourse to movement, are all that is required in the majority of simple fractures to effect a good result in the function of the limb, I am bound to admit that in many cases the disability is undoubtedly very great and often permanent. In simple fracture of the patella, for example, a certain loss of function in some instances may be great and should necessitate in suitable cases an open incision and wiring together of the fragments. Particular forms of fracture of the long bones are now dealt with by operative procedure. Severe displacements of the fragments in so-called simple (or closed) fracture, and cases in which it is quite impossible by manipulation to replace exactly the fragments in their original position, are now subjected to free exposure by an incision, and the fragments brought together by suture or screws, or by some other method of keeping them in position. The risk of such operations must be restricted to these particular instances, and as for the position of the fragments, which renders them justifiable, skiagrams are now taken of the limb from two points of view. Complete restitution, and not merely union in any position, must be arrived at, whether by mechanical means, as in Colles' fracture, or by operation, as I have said, in the more difficult fractures."

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY

No. 459. WEDNESDAY, AUGUST 14, 1901. Vol. XVIII. No. 17.

CONTENTS.

PAGE

*Chapters from the Teaching of Dr. Frederick Roberts. A PRACTICAL OUTLINE OF "CASE-TAKING," WITH COMMENTS 257

*Two Clinical Lectures on Intestinal Ulceration. By C. R. B. KEETLEY, F.R.C.S. Lecture II 265

*Intussusception and its Treatment. By D'ARCY POWER, F.R.C.S. 268

Treatment of Cancer of the Tongue. By J. BLAND-SUTTON, F.R.C.S.... ... 272

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

CHAPTERS FROM THE TEACHING OF DR. FREDERICK ROBERTS.

A PRACTICAL OUTLINE OF "CASE-TAKING," WITH COMMENTS.

THE essential value of systematic "case-taking" is self-evident, and is generally acknowledged in theory. As a means of clinical instruction and training its usefulness cannot be over-estimated from various points of view; and there is no more important part of the duties of a medical practitioner than to be thoroughly conversant with some plan for conducting the interrogation and examination of patients, so as to be able to do this in an intelligent and methodical manner, and at the same time with rapidity, while aiming at clearness, thoroughness, and conciseness. At first most students find considerable difficulty in "taking a case" properly, and they need for a while competent guidance and supervision. Of course much will depend on the nature of the particular case, and it is best to begin with those which are simple and straightforward. With adequate practice and attention, however, the process becomes more and more easy, even in relation to cases which present various degrees of complication. Many fail to reap due advantage from case-taking owing to a want of perseverance and energy. If the process has only been learnt in a perfunctory way, its results can only at the best be unsatisfactory, and may be decidedly mischievous and misleading.

At the outset it is most desirable that the clinical student should obtain some intelligent and comprehensive idea as to the meaning of "case-taking," and the course of procedure which it implies. It will be at once obvious that the particular points or details to be investigated in individual instances must present great variety; but here I merely propose to give a general outline of the plan which I am accustomed to follow, and to explain briefly the intention of its several stages. The points to

Digitized by Google

be noted and studied may be grouped under six primary divisions or stages, namely:

I. Personality and general history of the patient.
II. Family history.
III. Previous health, and past illnesses or injuries.
IV. Present illness.
V. Present state.
VI. Progress and termination.

For purposes of training it is well to begin by going through these several stages systematically and fully, in the order indicated, without reference to the actual nature of the disease. Subsequently, as the student gains experience, he will learn to direct his inquiries to the points of more immediate significance in relation to a particular case. If once by repeated practice familiarity with case-taking has been established, and it has become a habit, so to speak, its application in individual instances is natural and easy, and comes without conscious effort. Of course, in an ordinary way the medical practitioner has not the time as a matter of routine to write out a detailed account of every patient, nor is it necessary, but if properly trained he as a rule almost instinctively goes through the process in his own mind; and when required he should be thoroughly competent to conduct such inquiry and personal investigation as may be called for in the more important, interesting, or difficult cases which come under his observation. With regard to the usual run of his patients, moreover, he is already possessed of much of the information which he might otherwise have to elicit. During the process of education and training it is an excellent plan for the student, after taking a case, to give a brief *resumé* of its principal features, etiological and clinical. Later on, as his knowledge enlarges, he may with great advantage "write a commentary," discussing its diagnosis, prognosis, and treatment. "Clinical charts" and "outline diagrams" are now much employed to record various phenomena, such as temperature, pulse, respiration, weight, etc., and also to present graphic representations, where these can be used advantageously. Undoubtedly these are valuable aids in case-taking if properly employed.

As a preliminary to case-taking in hospitals, it is a common custom to ask the patient—(1) How long have you been ill? (2) What do you complain of? This is a good rule in an ordinary way, for we thus obtain information at once as to whether we have to deal with a case of sudden, acute, or chronic onset and duration; and will probably receive useful hints as to the class of disease we have to investigate, or at any rate as to the seat of the trouble, and the organs or systems which happen to be affected. At the same time the replies to these questions must be accepted with due reserve and consideration, otherwise they may seriously mislead, as will be pointed out presently. I now proceed to discuss the several primary headings in the order already enumerated

I.—Personality and General History.

The points to be investigated under this head refer to the past life as well as to the personal conditions and present surroundings of the patient, and they are often of essential importance from an ætiological aspect, and in their relation to diagnosis, prognosis, and treatment. Speaking generally they are definite enough, but their bearing upon individual cases presents great variety, and this part of the investigation affords ample scope for the intelligent application of a sound knowledge of general and special etiology. As a matter of routine the first step is to note the *name*, *sex*, *age*, and, under special circumstances, the *race* or *nationality* of the patient, which represents the more obvious and prominent facts relating to the personality of the individual. At the same time the *date of admission* into hospital or *when first seen* is recorded. The other points to be inquired into are concerned with the patient's social status and relationships, environment, or surroundings, mode of life and habits, and the chief of them are as follows:

1. *Whether married or single? Number of children.*—Of course it is not necessary to ask these questions, as a rule, in the case of very young subjects, at any rate in this country, but the fact that very early marriages and their consequences do sometimes take place must not be entirely forgotten. It must further be mentioned, as regards females more particularly, that, although single, they may be to all intents and purposes in the same relations as married women, and this fact it may be important to ascertain from an ætiological or diagnostic point of view. In this connection it is usual and convenient to inquire

Digitized by Google

whether there are any children, their number, and in some instances to note the rapidity of child-bearing.

2. *Social position and general mode of living.*—In certain cases it may be well to make a general reference to the social position of the patient, if it is not already known, and at the same time to note anything specially striking as regards the ordinary routine of social life and habits, particularly when dealing with those who are in affluent circumstances, or go much into "Society." The more important individual factors which are usually inseparably associated with the social status of a patient should then be separately discussed and inquired into. It may be well to observe that a sudden or rapid change in social position, especially "coming down in the world," may have a decided influence from an ætiological point of view.

3. *Residence and environment.*—It need scarcely be remarked that another point in case-taking which should be noted down as a matter of routine is the address of the patient, for the mere purpose of subsequent communication if required. The investigation as regards residence is often a very simple matter; but, on the other hand, it not uncommonly opens up many important considerations, not only in relation to the residence itself and its internal economy and arrangements, but also with regard to climate, effects of season, soil, aspect, elevation, amount of sunlight, general surroundings, sanitary conditions, and other matters. Hence much latitude is left for inquiry in these various directions, which individual cases will suggest. It is not uncommonly necessary to find out whether a patient has at any time resided abroad, and especially in unhealthy climates or malarial districts, and if so, where, and for what period? Ordinary changes of residence may also be worthy of note, as from the country to a large town or city, or from one locality to another, with reference to their effects upon the general health, or in relation to particular complaints or symptoms.

4. *Occupation.*—Under this head it must be mentioned that the want of any rational occupation or employment ranks as one of the causes of not a few complaints or ailments, and is therefore not uncommonly worth noting. There are various factors which tend to produce disease in relation to particular occupations, and these must be borne in mind in connection with this part of the investigation. An employment may be harmless enough, or even beneficial in itself, but becomes injurious because it is carried on for too many hours, or under unfavourable circumstances or conditions, and these are points which must also be duly noted. Not uncommonly a patient has followed more than one occupation during his lifetime, and it may be important to inquire into this matter, especially if there is reason to believe that some previous employment has entailed heavy physical work or strain. It is often well to ascertain whether an individual has served in the army, navy, or reserved forces, as the training connected therewith, while as a rule beneficial, may be decidedly injurious from an ætiological aspect when carried to excess, or in the case of unsuitable subjects.

5. *Exercise.*—Apart from occupation, it is often of much consequence to inquire into an individual's habits as regards the amount and kind of exercise taken, and whether it is regular and systematic, or irregular and spasmodic. A large number of persons suffer from want of or deficient exercise, leading more or less a sedentary and indolent life, or from lazy and luxurious habits generally. On the other hand, excessive exertion, either habitually or at intervals, in connection with athletics, various games, racing, swimming, field-sports, cycling, and other forms of voluntary effort, must be clearly and positively recognised at the present day as a potent factor in developing certain morbid conditions, and this aspect of a case must not be overlooked. The immediate personal training required for some of these exercises may also be decidedly injurious. More or less intimately associated with the question of exercise is that of whether the patient is sufficiently out of doors, and gets a due amount of fresh air; or is exposed to the strong sun and to weather of all kinds; or leads an indoor life, seldom if ever going out.

6. *Food and Drink.*—Questions relating to diet, and to the usual habits as regards feeding, naturally constitute an essential part of case-taking in every instance, but the points to be noted vary widely in different cases. The evils resulting from privation on the one hand, and over-indulgence on the other, are but too familiar; but there are many other matters which often require investigation as regards the quantity and quality of food taken, the fre-

Digitized by Google

quency and regularity of meals, the habit of bolting food, insufficient mastication, and other common errors. Habitual abuse of condiments may be an important point to ascertain in relation to diet. Inquiry as to drinks opens up the whole subject of the use and abuse of alcoholic beverages. This part of the investigation is as a rule simple and straightforward enough, but it is not always safe or reliable to take an individual's assertion of temperate habits as strictly correct, for with not a few persons the expression has a somewhat elastic meaning, and it is not uncommonly necessary to inquire into details, such as the kind and amount of stimulant taken; whether with meals only or at other times; if two or more kinds at the same meal; and, in the case of spirits, whether taken strong or diluted. Moreover, it may require very cautious and diplomatic investigation to detect indulgence in alcoholic excess, especially secret drinking, when this habit is suspected but not acknowledged. Abuse of tea or coffee is another well-known cause of certain disorders; while even over-indulgence in water, especially iced water, aerated waters, so-called "temperance drinks," or hot water—a "fashion" not uncommon at the present day—are errors not to be forgotten or overlooked when investigating many cases. Further, it is a familiar fact that water, as well as milk and other kinds of food, is frequently the channel by which various morbific agents enter the system, and this point must be prominently borne in mind in relation to diseases which may be thus originated.

7. *Special Habits.*—The chief causes of disease to be inquired into under this head are smoking, snuff-taking, and the frequent or habitual and often excessive employment of various medicinal agents, such as strong aperients, soporifics, anodynes or antineuralgics, and tonics. The pernicious habit of using subcutaneous injections of morphine, cocaine, or other agents, must never be forgotten at the present day in cases which are not clear, and where there may be reason to suspect any such habit, though its detection is by no means always easy, and may require much tact. Smokers, again, have often anything but a definite idea as to the meaning of moderation, and their statements cannot be accepted unreservedly. It may therefore be desirable to ascertain approximately how much they really do smoke; if strong or weak tobacco; and whether in the form of pipes, cigars, or cigarettes. The practice of opium-smoking rarely calls for notice in this country, but it must not be forgotten in countries where the habit is prevalent.

8. *Clothing. Personal cleanliness. Baths.*—It must suffice to mention these factors, which often have an important ætiological relation to particular diseases, as well as to individual cases. They should always receive due attention, and may call for more or less careful and methodical investigation. Details with regard to clothing are often of much consequence, and an intelligent knowledge of the causes of disease which belong to this head will suggest what points to inquire about in particular instances.

9. *Temperament and Disposition.*—It is always well to take a note of the so-called "temperament" of a patient, should it be in any degree pronounced or typical, though it often has no definite ætiological or clinical significance. This is generally described under the "present state," but it really forms an essential part of the personality of an individual, and may be conveniently referred to under the general history. The student should be familiar with the more characteristic features and morbid tendencies of the chief temperaments usually recognised, which are severally named *sanguineous*, *lymphatic*, *bilious*, and *nervous*. In relation to the last-mentioned system it is often desirable to ascertain whether a person is predisposed to become low-spirited and despondent or melancholy; to brood over and exaggerate unpleasant experiences, real or imaginary; to be unduly anxious or worried over trifles; to be easily excited; or to present rapid changes in mood and spirits. In other words, the clinical investigator has to seek for evidence of habitual irritability, unstable mental equilibrium, or a low tone of and want of proper control over the central nervous system, especially the higher nerve-centres, conditions which have potent ætiological relations.

10. *Mental causes.*—In addition to what has been mentioned under the preceding heading, there are certain definite causes of disease directly affecting the brain, and indirectly influencing other structures, which not uncommonly need to be inquired about in relation to the general history of a patient. They include principally excessive study or other kind of mental work; difficulties or irregularities as regards sleep; definite anxiety and mental strain of various kinds; prolonged grief; and the

Digitized by Google

worries so often associated with domestic life, business, social functions and aspirations, politics, and other aspects of modern life. These factors are not uncommonly combined in various ways in individual cases.

11. *Idiosyncrasies.*—It is a well-known fact that individuals are affected by certain kinds of food, or by drugs, in a peculiar manner, and it may be worth while to investigate this point in certain cases. Patients not uncommonly affirm, moreover, that they are particularly liable to be affected injuriously by special causes, or that these act upon them in an unusual way. It must not, however, be taken for granted that such statements are always correct.

12. *Sexual causes.*—This class of causes must not be overlooked, though it is obviously not necessary or desirable to make any reference to them in the investigation of the ordinary run of cases. Under what circumstances an inquiry in this direction is called for must be left to individual judgment and discretion, but it must not be shirked when really required. Masturbation and venereal excesses afford but too familiar illustrations of the errors here referred to.

II. Family History.

In the process of case-taking some clinical teachers and writers place the "family history" first in order, and logically this is correct, but it does not make any material difference, and personally I prefer bringing it in here. What is more important is to have some intelligent idea of the objects which this part of the inquiry has in view, and, speaking generally, what we have to aim at is to ascertain whether there is any hereditary taint or tendency to a particular disease, or other family peculiarity having a pathological significance. While primarily and mainly fixing attention upon parents, brothers and sisters, and children, it must not be forgotten that under certain circumstances it is requisite to inquire respecting grandparents, or even generations further back, if the information can be obtained; and also concerning collateral relatives on both sides—uncles, aunts, and cousins. When taking a complete history of any case it is customary to note any deaths in the family, with their causes, and the ages at which they occurred, as well as the state of health of its living members; but how far these details may be called for in individual instances must be left to the discretion of the investigator. The chief facts which investigation of the family history may reveal are—

1. A tendency to death at an early age, or about a certain period, in several members of the family.

2. A predisposition to premature degeneration or senility, the degenerative changes sometimes affecting many structures, though they may be revealed in various directions, and are not necessarily identical in different generations.

3. A definite history of the more or less wide prevalence of certain special complaints or conditions, usually regarded as distinctly hereditary, either on one or both sides of a family. At the present time there is a disposition in the medical profession to question whether some important diseases, usually regarded as strongly hereditary, actually belong to this category, but this matter cannot be discussed here. It may be affirmed, however, that the laity have notions as regards heredity which are certainly by no means always in accordance with fact.

4. A history of some pernicious habit in one or both parents, or it may be in more than one generation, *e. g.* intemperance, likely to lead to disease in the offspring.

5. Descent from parents nearly related to one another, very young, or of very unequal ages; or from those who are delicate and weakly, especially in the case of the mother, who often is the victim of repeated or persistent debilitating influences, prominent amongst them being excessive child-bearing and nursing.

6. A family tendency to be attacked by particular diseases, such as some of the infective fevers, or to have them severely or in a very malignant and fatal form.

The preceding summary is merely intended to give the student a few practical hints to guide him in his inquiry with regard to the family history of a patient. The information thus derived is often of great value, but at the same time some degree of caution is necessary in forming conclusions founded thereupon. It may happen that members of a family or of different generations are exposed to the same cause or causes of a particular disease, not forgetting infection, and what appears to be a hereditary complaint is not really so. This remark

Digitized by Google

applies conspicuously to tuberculosis. On the other hand, it may not always be easy to obtain a direct history of a hereditary taint, of the existence of which there is a strong suspicion, or to elicit positive information of other kinds. In doubtful cases personal examination of the parents or of the patients themselves will sometimes clear up this point, especially in the case of congenital syphilis, and a history of repeated miscarriages may also help in indicating the presence of such a taint.

III. Previous Health and Past Illnesses or Injuries.

This portion of a patient's history is often of essential importance, and its bearings in a particular case are by no means always so clearly or intelligently recognised as they might be. Taking a comprehensive view, the chief factors to be noted may be thus indicated:

1. The habitual state of the general health and condition. Not uncommonly there is a history of "delicate health" from early life, or for a more or less prolonged period, or of anæmia; or that the patient is always "ailing," though not suffering from any definite complaint. It must be remembered, however, that neurotics and hypochondriacs will frequently give a record of prolonged illness which has no real foundation in fact. Of course, in a large proportion of instances there is nothing particular to note under this head, the majority of people enjoying average health, and not a few having been in excellent or even robust condition previous to the time when they come under observation as patients. It does not follow, however, that they have been really sound, for it is not at all uncommon to meet with cases in which a serious morbid change has been going on insidiously and unconsciously for a considerable time, until some slight disturbing cause finds out the "weak spot"—a fact not always sufficiently appreciated. Acute illnesses often attack people previously in good health and condition, and sometimes they are almost too vigorous or abnormally plethoric.

It may be observed in this connection that it is well to make it a rule to ascertain how the normal functions are habitually carried on, especially as regards digestion, action of the bowels, and menstruation. It is extraordinary what a number of people suffer more or less from constipation, and this has an important bearing upon a considerable proportion of cases of different kinds in ordinary practice. Sometimes it is requisite to make inquiry also with regard to the urinary functions.

2. A history of known past illnesses and their effects. Among these may be specially mentioned—

(*a*) One or more definite illnesses or diseases, generally acute, which have left behind either more or less obvious permanent lesions, injurious in themselves or likely to produce further mischief; a susceptibility to some other complaint; an increased predisposition to a return of the same affection; or a direct liability to further morbid processes of a particular nature.

(*b*) The occurrence of paroxysmal attacks or "fits," coming on at intervals of various durations, and either regular or irregular, indicative of well-recognised complaints, such as ague, asthma, angina pectoris, gout, hepatic or renal colic, epilepsy, or migraine.

(*c*) An abnormal liability to functional disorder, or even to actual disease of a particular organ or system. As illustrations may be mentioned a history of susceptibility to "cold," with bronchial attacks; of dyspeptic or hepatic disorders; of diarrhœa easily induced; of cardiac disturbances; of neuralgic and other nervous complaints; or of a tendency to certain cutaneous affections.

3. The previous existence of some chronic and permanent morbid condition, of which the patient may or may not be aware, and which is liable to set up some secondary lesion or accidental complication—as exemplified by chronic phthisis, throat complaints, disease of the cardiac valves or orifices, arterial changes, varicose veins, gall-stones, cirrhotic liver, hernia of different kinds, and chronic renal disease. It may also be mentioned here that a history of a symptom having persisted for some time, or being prone to recurrence, although not attributed to any definite complaint, may be of much significance and worthy of note in individual cases, and it is often even directly injurious in itself; for example, violent cough, sickness, various morbid discharges, or hæmorrhages.

4. A history of some condition which has necessitated operative interference. In this relation it may be desirable to make special inquiry as regards such complaints as pleurisy or empyema, glandular diseases, nasal and throat affections,

Digitized by Google

diseases of bones or joints, and other conditions which individual cases may suggest.

5. A history of injury, more or less serious, which has either occasioned direct and obvious effects, subsequently originating other lesions; or which, although it has not produced any evident immediate consequences, has laid the foundation for some complaint which in course of time reveals itself, as is exemplified in the relation between railway accidents and certain nervous affections.

When taking the part of a patient's history just outlined, it may be necessary to enter into various details, such as the time of life at which an illness occurred, its severity and duration, the treatment adopted, whether recovery was complete or if it left after-effects, and so on. It is particularly necessary to guard against being misled by the expressions and names of complaints which patients often use, without knowing their real significance, such as pleurisy, congestion of the lungs, brain-fever, etc. In cases of doubt inquiry as to symptoms and other points may help materially when we have to deal with an intelligent patient; or personal examination may afford corroborative evidence as to the truth of a statement. Moreover, such examination may reveal signs of some previous illness, or of some pre-existing morbid condition which has developed insidiously, and of which the patient is quite unaware. Again it not uncommonly happens that individuals do not realise the importance of complaints from which they have suffered, or they have forgotten all about them, and a history can perhaps only be elicited by close inquiry. In the case of young subjects, or when an illness of which the remains are more or less evident has occurred during early life, an endeavour must be made to obtain information from parents or others acquainted with the facts, but it may be impossible to learn anything definite about such illness, although we may be sure it has happened. It must not be forgotten that persons sometimes intentionally conceal or deny the fact that they have had some special disease, particularly syphilis, and then it must be left to the intelligence and tact of the investigator to clear up matters in suspected cases.

IV. Present Illness.

The history of the illness from which a patient suffers when he comes under observation is as a rule readily obtained if the inquiry is carried on intelligently and methodically, but sometimes caution is necessary in accepting statements offered under this head, as has already been intimated. The main points to be determined in relation to the present illness are—

1. *Its duration.*—In most instances approximate information on this matter is sufficient for all practical purposes, but in dealing with certain affections, especially some of the acute specific and inflammatory diseases, it is important to endeavour to fix as accurately and definitely as possible the date of the onset of the illness, or even the exact hour sometimes. In not a few cases the invasion of a disease has been so gradual and insidious that no reliable conclusion can be arrived at as to its duration, and, as already stated, such disease may have existed for a long time without the knowledge of the patient, its presence perhaps being only revealed by some accidental occurrence. Under these circumstances incorrect information will probably be given on this point, against which the clinical investigator must be on his guard. As bearing upon the duration of an illness, it may be helpful to ascertain when the patient gave up work or took to his bed.

2. *Its immediate exciting cause or causes.*—The line of investigation already discussed is intended in the main to find out whatever causes or conditions may have had any indirect influence in originating the disease or illness with which the practitioner has to deal, these being usually classed as *predisposing*. Now he has to inquire more or less carefully as to the more direct or *exciting* cause or causes which have brought it about. Often this part of the investigation is simple and easy enough, and not uncommonly we are able to fix upon one definite cause without any difficulty, but there may be more than one, perhaps several, acting in concert, which it may be important to recognise. It sometimes happens, however, that no positive cause can be traced, even though we know it must actually exist, as in the case of infection. This is one of the causes of disease which is particularly liable to be intentionally concealed under certain circumstances. On the other hand, patients or their friends may attribute an illness to some ætiological factor which is altogether wrong or imaginary.

3. *Its mode of onset or invasion.*—Under this

Digitized by Google

head it is sufficient to note, by the well-known terms in common use, whether an illness has been sudden, acute, subacute, or gradual and chronic in its onset.

4. *Its initial or early symptoms, and clinical history up to the time when the patient is first seen.*—Here more or less description is usually required, and many opportunities are afforded for individual judgment and tact in bringing out and weeding the more important facts, without being too prolix or indefinite. Much depends upon the knowledge which the investigator possesses with regard to the clinical history of the various diseases usually met with, including the so-called *premonitory* or *precursory* symptoms, or *prodromata*, which not uncommonly give intimation or warning as to the approaching development of a particular complaint, that may not have fully declared itself when the patient comes under observation for the first time.

5. *The treatment* adopted, if any, and its results.—This point is sometimes worthy of consideration, but it is always necessary to be particularly cautious in accepting or founding any conclusions upon the statements made as to what has been done in the way of treatment, and especially as to the supposed effects of the administration of medicines by the "doctor."

V. Present State.

To "take the present state" of a patient must be regarded as the essential and indispensable part of clinical investigation, for however much the several stages just discussed may be neglected or ignored, it is manifestly impossible to deal with any case without having some knowledge of at any rate the more prominent symptoms complained of or observed, and even in the ordinary routine of medical practice it may be assumed that at least a show is made of carrying out whatever kind of examination such symptoms suggest or demand. The clinical student must realise fully at the outset that not only is this by far the most important part of the investigation, but that it presents many and various difficulties, and it must be emphasised here that only by long-continued and special training can its details be intelligently and satisfactorily mastered. Therefore he must practise in as large a number and variety of cases as possible. At first it will be well for him to go through the whole process of taking the "present state" so as to gain familiarity with its details, investigating every organ and system even though it is not obviously affected, but he will gradually learn by experience how to direct and limit his inquiries, according to the nature of the case with which he is immediately concerned. The intention of the procedure is to obtain a systematic, orderly, complete, but at the same time concise and rational description of the clinical phenomena observed or elicited by a personal examination of the patient, not only for the purpose of immediate diagnosis, but also for future record. Among the more common mistakes to be guarded against are a want of any system or method; a lack of discrimination and appreciation of the value and importance, relative or absolute, of the phenomena to be observed and noted; and unnecessary diffuseness or verbosity. Many supposed symptoms are often described very fully which are of little or no consequence, while others of real significance are either overlooked and ignored completely, or referred to only casually.

When describing the "present state" at length, it is customary and convenient in the first place to take note of *general symptoms* or *conditions*, and of *local external phenomena*; and afterwards to discuss more or less fully the phenomena connected with the *several organs and systems of the body*, including amongst them those that are brought out by the various methods of skilled examination sketched out in a previous article in this Journal. The consideration of the details of this part of case-taking is obviously altogether beyond the purpose of this communication; and it must suffice to say that in actual practice the extent, range, and course of investigation required differ vastly in different cases. In some instances it is a very simple matter, the clinical phenomena being few in number, well-defined, and limited to one organ or system; in others it has to be more or less elaborate, and in complicated cases it is by no means easy to give a description which shall be sufficiently comprehensive and lucid, and at the same time comprised within a moderate compass. It is a good rule to try to obtain some clue at the outset as to the region of the body or the organ to which the prominent symptoms point, which is often a useful guide as to the line of inquiry to be

Digitized by Google

pursued. When taking fully a "present state" it will be well always to make a general inquiry about every system, and if there is nothing to be recorded, to sum it up as "normal." No useful purpose can be served as a rule by enumerating a series of symptoms merely to state that they are absent, though there are cases in which the absence of a particular symptom may be important, and therefore worthy of note.

VI. Progress and Termination.

It is often requisite to watch cases more or less carefully during their course, and to take notes from time to time of the progress of events. In some instances this is absolutely essential for the mere purpose of diagnosis, which at first it may be impossible to arrive at, or it is uncertain, and requires to be corrected or corroborated by subsequent observation. The observation of the effects of a particular line of treatment may afford valuable information from this point of view. But, further, there are many cases of well-recognised diseases, especially those of an acute nature, the clinical course of which has to be systematically studied, with the results of treatment, and particular attention is required when *complications*, *sequelæ*, or other secondary developments are likely to arise, of which due note must be taken. Some complaints are liable to *relapses* or *recurrences*, the occurrence of which must also be recorded. In order to complete the account of a case, its *termination* must be stated, whether in death, recovery, or the establishment of permanent and chronic disease, adding such details as may seem called for in individual instances. This concludes "case-taking" so far as relates to clinical medicine. Should death occur, and a *post-mortem* examination be made, this ought to be carried out in the same systematic and complete manner, but the details relating to such examination come within the scope of the pathologist, and are beyond the province of this communication.

Digitalis.—Having had his attention called to a case of acute delirium and mania apparently due to the use of digitalis, Hall looked up the literature and finds the subject treated in a monograph by Duroziez, who reports twenty cases.—*Journ. A. M. A.*, July 13th 1901

TWO CLINICAL LECTURES ON INTESTINAL ULCERATION.

Delivered at the West London Hospital.

By C. R. B. KEETLEY, F.R.C.S.,
Senior Surgeon to the Hospital.

Lecture II.

Gentlemen,—The diagnosis of the cases in which perforation takes place is, of course, very important. When an intestinal ulcer perforates simply into the peritoneal cavity it generally causes the patient to collapse unless the perforation is very gradual. As a matter of fact intestinal perforation often takes place slowly. Everything depends on whether the peritonitis is a generalised or a localised one. It also occurs sometimes that the perforation leads into the subperitoneal tissue, because over the ulcer there is no peritoneum; or adhesions are formed before perforation. It would take too much time to enter now into the question of peritonitis and the symptoms of it, and possibly you are as familiar with it as I am, as, unfortunately, it is such a common thing. One also has to remember that perforation may occur into the bladder, as in the case of an unfortunate patient I referred to last time.

Treatment.—I should now like to say a little more about the general principles of treatment which is not necessarily operative. First of all with regard to innocent ulcerations in general; rest in bed is a good thing in itself. Then the patient should have a purely milk and beef-tea diet, or something corresponding to it. Some people say that the salicylate of bismuth and also salicylate of soda are good things to give internally. Salol is also sometimes given. In fact, very much the same kind of treatment is followed as in acute and chronic enteritis. One thing is certain, namely, that meat should be avoided in cases of ulceration of the intestine, because the fibres of meat seem peculiarly irritating. One notices this also in colitis. Unfortunately most of the antiseptics seem to disappear into the blood before getting far down the intestine. However, as I need hardly say, the large intestine can be treated from the rectum, and in almost all forms of ulcerative inflammation of the mucous membrane of the large intestine liberal enemata given very gently and patiently are beneficial. There is no doubt about

Digitized by Google

these large enemata reaching the whole length of the large intestine, because I have often seen, in cases where there has been a fistula into the cæcum, that fluid has come out at the fistula when it has been injected into the rectum. A favourite salt for all ulcerations of the large intestine and the one which seems to do the most good is nitrate of silver; I am speaking now of innocent ulcerations. Malignant ulcerations are much better not irritated with strong solutions. I think it would be well worth while in non-malignant cases to try one of the new silver salts which one reads about, such as argentol and argentamin, which are said to penetrate into the mucous membrane without coagulating albumin. The strength of the nitrate of silver used is generally half a grain to the ounce. In order to get the injection to reach all parts of the large intestine it is necessary to have the patient with the hips high. Boracic acid sometimes is used in a similar manner. Another thing which undoubtedly does good in some of these obstinate chronic ulcerations of the large intestine is change of air. An old lady had symptoms of inflammation of the mucous membrane of the large intestine for years, and also some slight indications of ulceration. There was no reason to suppose she had stricture. She changed her residence about a year ago and went to a rather bracing place, and it is wonderful how much better she has been. It is not at all unlikely that the catarrhs which lead to these ulcerations in old people are sometimes connected with bad teeth, and consequent improper mastication. If ulceration once commenced the bolting of quantities of fibrous meat tissue would be the thing to keep it up.

Sometimes strangulated hernia which has been spontaneously recovered from will cause stricture. An old man came into the hospital very ill with strangulated hernia. He was so bad that he was not fit to take any general anæsthetic. I used a little cocaine, which was rather a sham; I practically operated upon him without any anæsthetic at all. He died, and his intestine was found to be greatly narrowed in different places. Most probably some inflammatory change had caused this. A great extent of the intestine was made narrow by the existence of these strictures.

Unfortunately most *tumours* of the intestine are malignant. Such tumours as papillomata, adenomata, and lipomata are found, especially in the lower end of the intestine—that is to say, the rectum. I removed a fibroma from a middle-aged lady's rectum a few years ago. Naturally the presence of the tumour near the anus alarmed us at first, but it turned out to consist only of fibrous tissue, and she has never had any recurrence. It was simply a hard mass as big as half an egg on one side of the rectum.

Tubercle not only causes ulceration, but it sometimes causes what at all events has the clinical appearance of an intestinal tumour. A boy came in here some years ago with quite a large solid, well-defined tumour on the right side of the abdomen, on a level with the umbilicus. On putting the left hand under the flank, and pressing down with the right, the tumour could be felt to occupy the whole depth of that side of the abdomen. The question, of course, was whether it was kidney, or whether it was a tumour of the intestine. Had the patient been older it would have also been a question whether it was gall-bladder or liver. I made an opening into the peritoneal cavity, and found the tumour attached to the anterior as well as to the posterior wall of the abdomen, near the kidney. I could feel the lower end of the kidney and the tumour moving with it, so it was really attached to the kidney itself. But I also discovered in this examination that it contained intestine—that either it was surrounded by intestine, or that it was made up of intestine. So I made an incision down into the tumour itself, and found it really was a mass of intestines glued together by tuberculous inflammation. Through this second incision I separated the intestines, taking care not to go into the general peritoneal cavity, went down to the kidney, and discovered three or four distinct collections of tuberculous pus. I cleaned them out gently, and put in some iodoform. I did not confidently expect a good result. However, the boy apparently got quite well, and all trace of the tumour disappeared. Some years afterwards he died of a kind of marasmus, which was probably due to tuberculous disease of the mesenteric glands. At the time I operated on him I found he had numbers of enlarged mesenteric and retro-peritoneal glands.

Now we come to the tumours which are of most importance, namely, the malignant ones. They are either epithelioma or sarcoma. The form of

Digitized by Google

epithelioma which affects the intestine is generally what is known as cylindrical epithelioma. As you know, the rectum is the region which, above all others, is affected by it. Indeed, one may say generally that malignant tumours usually affect the lower part of the large intestine—either the rectum or the sigmoid flexure, or some part of the colon not far off. However, one of the largest and worst cases I have seen affected the splenic flexure of the colon. It began in the splenic flexure of the colon, and ultimately absorbed the whole of the omentum, and also affected a large coil of small intestine. It was one of the first intestinal operations I did. It was an extremely movable tumour, notwithstanding that it was so very large, and obviously the man was bound to die if it were left. I had to excise the whole of his descending colon, all the splenic flexure of the colon, and a good part of the sigmoid flexure of the colon. I also had to excise a large piece of small intestine, and cut off all the omentum from the stomach. This operation took three and a quarter hours. The patient only lived twenty-four hours after the operation. I learnt something from this case, namely, what the intestine and abdomen generally can stand, both pathologically and operatively. The man was anæmic and weak, and to the last degree emaciated through the growth. It was so movable that it tempted the surgeon to interfere, and when once he begins such an operation he must go on.

The other form of malignant tumour which is not infrequent in the intestine is sarcoma, and that affects the small intestine rather than the large. This, however, only applies to primary sarcoma of the intestine. Still, I myself think that if one case were taken with another, and the cases of sarcoma beginning in the pelvis outside the intestine altogether, and ultimately infecting the intestine, or causing external stricture, we should find that sarcoma was more common, like cancer, as a tumour of the large intestine than of the small intestine. I have only had the opportunity of operating on one case of sarcoma of the small intestine, and that was quite a type. The patient was a young girl of thirteen or fourteen years of age, who had symptoms of what might be called subacute intestinal obstruction. She was very thin, and there were two tumours to be felt, quite distinct and separate. The lower tumour was quite fixed, and it appeared that it had been the first of the two to present itself and be observed. The other tumour was discovered afterwards, and was more movable; it could be shifted up and down freely. On finding this to be the case I made an incision over the lower one, and then I easily got the upper one down under the incision. It turned out to be more superficial than the lower one. It was evident when I got this tumour into view and to touch that it was inside a piece of large intestine. The next thing I noticed was that the cæcum and the appendix had disappeared; they did not occupy their proper place. Then I guessed what really was the matter. This was a tumour of the intestine, which had caused intussusception of itself, the ileo-cæcal valve, and the cæcum into the ascending colon. I made an incision into the large intestine, and found the intussusceptum projecting into it. Having cleaned away some fæces, I put on a large clamp on the base of this intussusceptum, and cut it away. I united the parts by a modification of what is known as Maunsell's operation. It is a capital operation and should be known to you because it requires no special appliances—nothing but ordinary sewing needles. In this case I thought it would be well to remove the second and more fixed tumour on a future occasion, as, to take it away, I should have had to remove some of the vessels which supply the intestine where I had done the enterorrhaphy, and I did not like to run the risk of causing gangrene there. When I proceeded to remove the remaining tumour, three weeks after the first, it had grown very much. The patient recovered, but with a fæcal fistula. She ultimately died of exhaustion and intestinal obstruction. The second tumour recurred, and grew rapidly to an enormous size. I may mention it was extraordinary that there was a retro-peritoneal tumour such as I have described, because it is not customary for sarcoma to infect the glands. When there is carcinoma of the intestine the glands in the mesentery do, sooner or later, become enlarged.

Sarcoma infiltrates the cellular tissue and makes a huge sarcomatous mass in which the intestine forms a mere canal. That is very well seen in cases of sarcoma of the pelvic organs. I operated upon a woman who had acute obstruction supervening upon something amiss with her pelvic

Digitized by Google

organs which had not been diagnosed. I found that her pelvis was full of a solid mass. I was not able to do anything radical, but I brought the sigmoid flexure into the wound and did a sigmoid colotomy. She was *in extremis* when I operated upon her, and she did not survive the operation twenty-four hours. Post mortem it was found to be a sarcoma. Mr. Alban Doran examined it. It had infiltrated almost all the cellular tissue of the pelvis. The upper part of the rectum would scarcely admit a quill pen.

There is one form of stricture of the intestine which has interested me a good deal, and that is congenital imperforate anus. Two old hospital patients of mine, one at least of whom is now a big lad, were operated upon in the way I shall explain to you, which is the way in which such cases should be operated upon. In the first place the child was laid on its face on a water cushion containing warm water. It is necessary to keep a newly-born child warm. The operation was done from behind. In each of these cases there was a little rudimentary distal part of the rectum, as is usual. The incision was made from the lower piece of the sacrum, as near as I could judge, along the left edge of the coccyx down to the anus. This divided the skin and fat. The edges were retracted, and then the gluteus maximus appeared to the outer side. There is another piece of muscle going from the margin of the coccyx, part of the coccygeus and of the levator ani. It is necessary to incise the latter muscles. Then one comes across a layer of fascia, and the great sacro-sciatic ligament, which may be cut if found in the way. Having gone through whatever fascia is met with, one ought to be able to find the bowel, which is forced down here by its great distension with meconium, however high up the obstruction may be. The bowel is a purple-blue object. It is necessary to secure every bleeding vessel of the slightest consequence all the way down to the bowel. Another thing to remember is that when you get hold of and empty the bowel you must never lose it again, because you would have a difficulty to rediscover it. It is best to seize it with forceps, or, if you cannot, put a suture through. It has got to be opened as low down as you can, and then you will immediately find quantities of meconium come away. Empty the intestine, and wash it out with warm water. It is very important to suture it to the neighbouring parts in such a way that it cannot slip back. And, last of all, if the case is one in which the anal end of the rectum is patent, connect the upper segment with the lower segment by two or three sutures. Having done that, you have provided a free passage for the fæces, and the child should be sent back to its mother. I have operated upon four, more or less in the above manner. In the two cases which died I omitted what I believe now to be essential. In one case I omitted to suture the bowel in position; it looked as if it would have remained all right. It did not, however, and when the child was brought back several days afterwards I had the greatest difficulty in finding it again, and the second operation really caused the child's death. The other child which died I kept in the hospital, and I am sure that the difficulty of keeping these infants warm and feeding them properly by hand is a serious thing. But when they are sent back to their mothers that difficulty disappears. In the case of the two children who lived I sent them back to their mothers, having tied every vessel. There was no necessity for dressings. The only immediate after-treatment required is frequent changing of aseptic or clean cotton wool. After a few weeks, and for a few years, at intervals, it is desirable to pass in forceps and open them gently to dilate the anus.

INTUSSUSCEPTION AND ITS TREATMENT.*

By D'ARCY POWER, F.R.C.S.Eng.,
Senior Surgeon to the Victoria Hospital for Children, Chelsea; Assistant Surgeon to, and Teacher of Surgery at, St. Bartholomew's Hospital.

MR. PRESIDENT AND GENTLEMEN,—I think that the object of this discussion will be promoted if we give, so far as lies in our power, an accurate survey of the cases of intussusception with which we are best acquainted. I have therefore drawn up the accompanying table of the cases which have been admitted to the surgical wards of the two hospitals to which I am attached—St. Bartholomew's and the Victoria Hospital for Children. The cases enumerated in the table were admitted during the ten years ending December 31st, 1900.

* Remarks made during a discussion on the "Treatment of Intussusception," in the Section of Diseases of Children at the Cheltenham meeting of the British Medical Association, on Wednesday, July 31st, 1901.

Digitized by Google

Table of Cases of Intussusception occurring at St. Bartholomew's Hospital and at the Victoria Hospital for Children, Chelsea, 1891—1900.

	Cured.		Died.		Total.		Grand total.	Total No. of surgical patients.
	M.	F.	M.	F.	M.	F.		
1891. St. B. H.	0	0	1	2	1	2	3	3,852
Victoria Hospital	1	0	0	0	1	0	1	349
1892. St. B. H.	1	1	2	0	3	1	4	3,750
Victoria Hospital	0	0	1	1	1	1	2	441
1893. St. B. H.	3	2	1	1	4	3	7	4,539
Victoria Hospital	0	0	2	0	2	0	2	479
1894. St. B. H.	0	0	1	1	1	1	2	4,453
Victoria Hospital	0	0	1	0	1	0	1	565
1895. St. B. H.	1	0	5	2	6	2	8	4,617
Victoria Hospital	0	0	1	1	1	1	2	537
1896. St. B. H.	1	0	2	0	3	0	3	4,475
Victoria Hospital	0	1	1	0	1	1	2	475
1897. St. B. H.	0	1	0	2	0	3	3	4,237
Victoria Hospital	1	0	0	1	0	2	2	491
1898. St. B. H.	2	0	3	1	5	1	6	4,236
Victoria Hospital	1	0	0	0	1	0	1	387
1899. St. B. H.	4	1	2	2	6	3	9	4,120
Victoria Hospital	0	0	2	0	2	0	2	151
1900. St. B. H.	2	0	3	0	5	0	5	3,754
Victoria Hospital	0	0	0	0	0	0	0	289
	17	6	28	14	44	21	65	46,197

Total recoveries, 23. Total deaths, 42.

30 cases presented points of especial interest as follows:

2 recovered spontaneously, one acute and one chronic case.

1 recovered from a triple intussusception.

1 died of ileus after reduction.

1 had intestinal obstruction due to adhesions after the abdominal section.

2 cases were too ill to operate upon.

1 case had such extensive adhesions that nothing could be done.

3 cases recurred repeatedly after apparent reduction by irrigation.

In 3 cases the intussusception was overlooked at the operation and was found post-mortem.

In 1 case there was no intussusception.

15 cases needed excision of the bowel; in—

7 cases an artificial anus was made; all fatal.

5 cases Murphy's button was used; four died, and one recovered.

2 cases Maunsell's operation was performed; both died.

1 case a circular enterorrhaphy was done; it died.

A tabulated statement, however, is but a dull thing, and I propose to render this one rather less dry by calling your attention to a few of its more interesting points. I hope in the first place that the story told by the table is quite accurate. All the cases which it contains came under the personal observation of my colleagues or myself; the notes were taken under the supervision of the house-surgeons, controlled, as is the custom at St. Bartholomew's Hospital, by the Surgical Registrar; the post-mortem examinations were conducted, and the records were made, by independent surgeons who had often no knowledge of the case during life. The observations are free, therefore, from any personal bias, and the table is consequently more reliable than one compiled from published cases.

The first result which appears from the table is the rarity of intussusception in London, as only sixty-five cases have occurred amongst 46,197 surgical cases admitted into the two hospitals in ten years. Indeed, the Victoria Hospital, which is entirely devoted to the reception of children, only received fifteen cases in 4164 admissions to the surgical wards. If this is at all a usual average no single surgeon can have had a very large experience in the treatment of intussusception, and there is consequently the greater need for us to lay our heads together on such an occasion as this to learn what we can of each other's practice and methods.

The second point of interest is the very distressing mortality which attends intussusception even by modern methods of treatment. Forty-two deaths and twenty-three recoveries is by no means a satisfactory conclusion even when every allowance is made for the severity of the disease and the tender age of the patients.

An examination of the cases in a little greater detail shows that thirty of the sixty-five cases presented points of especial interest. Two are said to have recovered spontaneously. One of these was acute, and occurred in a child æt. six years, who presented the characteristic signs and symptoms of intussusception, which disappeared in a short time. The process of cure was, I suppose, a simple resolution of a short invagination. About the second case I am more doubtful. It was that of a boy æt. five months who had suffered from symptoms of intussusception for a month before his admission to the hospital. No operation was performed, and he is said to have left the hospital in an improved

Digitized by Google

state. The surgeon who watched the case for a month made a diagnosis of chronic intussusception. But I have lately had a case under my care (the 'British Medical Journ.,' vol. i, 1901, p. 1401, case ii) which appears to give a more easy solution. In my patient, who was admitted for the relief of an acute intussusception, there was a marked sausage-shaped swelling below the right costal cartilage. This swelling might easily have been mistaken for an intussusception, though I was careful to point out to my dressers before operating that it appeared to be too freely movable for the ordinary tumour of intussusception. I opened the abdomen and found an invagination of the colon into the colon at quite a different part of the belly. The swelling felt before the operation consisted only of the cæcum thickened by chronic inflammation, and made more obvious by several enlarged lymphatic glands lying over the ileo-colic junction. The chronic intussusception, therefore, which is said to have recovered might very easily have been a similar case, the signs and symptoms of obstruction being due to a localised inflammation of the bowel.

An abdominal section was performed in one case where there was no intussusception, and the child happily recovered. The converse mistake occurred three times where the abdomen was opened, but the intussusception was overlooked and was only found post mortem. These cases are particularly instructive, for in each case the post-mortem record states that the invagination was large and well marked, and it is to be remembered that the operations were performed by thoroughly competent hospital surgeons well skilled in abdominal surgery. The mistake therefore is easy to make, and its occurrence should be especially borne in mind.

The following case exemplifies the vitality of some boys, and contradicts the received teaching that all multiple intussusceptions are "agonic" in origin. A school-boy æt. twelve suffered for four days from abdominal pain and vomiting, but without collapse or abdominal distension. The abdomen was opened, and three intussusceptions were found, one at the ileo-cæcal valve and two in the small intestine. None of them appeared to be acutely strangulated. They were all easily reduced, and the patient made an excellent recovery.

I am not now concerned with the pathological or scientific side of intussusception, and I should probably weary you if I examined in detail all the interesting points which the table reveals. I pass on therefore at once to the important question of treatment. I used to think it wise to try to reduce early intussusceptions by irrigation with hot saline solution passed into the rectum under a low head of pressure. A larger experience has taught me that this method is unreliable, and I am therefore at present a whole-hearted advocate of abdominal section at the earliest possible moment in every case of intussusception. My change of opinion has been largely brought about by the following cases and others like them:—(i) A male infant æt. three months passed some blood and mucus a few hours before admission. It looked ill and an intussusception was felt on the left side of the abdomen and through the rectum. An injection of milk and water caused the tumour to disappear and the bowels acted several times. The intussusception returned on the following day, and was again reduced by irrigation of the bowel. On the fourth day after admission it returned again, and could not be reduced. The abdomen was opened on this day, and a large ileo-cæcal intussusception was found extending as low as the rectum. It could not be reduced, and the peritoneum was torn in the attempt. The intestine was opened above the intussusception, emptied, and then sewn up again. The child died a few hours later, and a little more than four days after the first onset of the symptoms. It was clear at the post-mortem examination that the intussusception had never been completely reduced.

(ii) A male infant was admitted with symptoms of intussusception of thirty-six hours' duration. A well-defined swelling was felt in the left iliac region, which disappeared when the large intestine was filled with milk and water. The swelling returned in a few hours, but as the symptoms of obstruction persisted an abdominal section was performed. Some congested intestine was seen when the abdomen was opened, and this was assumed to be a reduced intussusception as no other tumour could be felt. The child died thirty hours after admission, and the post-mortem examination revealed a large intussusception extending downwards into the rectum and lying behind the distended small intestine which had been seen during the operation. It seemed clear from the state of the bowel that the intussusception had never been completely reduced.

Digitized by Google

(iii) A girl æt. five and a half months was admitted with symptoms of intussusception of nineteen hours' duration. Six ounces of warm saline solution were run into the rectum from a height of 16—24 inches, the child being under chloroform. The fluid was allowed to remain in the bowel for ten minutes, and after its expulsion the tumour had disappeared, and a finger passed through the anus felt a sharp edge a short distance up the rectum at the place where the end of the intussusception had rested. The symptoms returned rapidly three hours later, when the child again began to vomit and to pass blood and slime. The irrigation was repeated, but the tumour did not disappear though it became smaller. The abdomen was opened three and a half hours later, and the bulk of the intussusception was easily reduced without bringing the tumour into the wound. It was impossible, however, to reduce the invagination completely in this manner because the adjacent sacculi of the caput cæci were inflamed and united together by lymph. The ileo-cæcal angle was therefore brought out of the wound to complete the unravelling of the last half inch. The child bore the operation well, but it only lived twenty-five hours longer, dying fifty hours after the first seizure. The post-mortem examination showed that the intussusception was completely reduced, and that there was no general peritonitis. Two adjacent sacculi of the cæcum were adherent by the slightest trace of lymph, the caput cæci was firmly constricted, and the whole cæcum was congested and thickened by the products of inflammation.

(iv) A boy æt. fourteen months was admitted with the symptoms of an intussusception of six days' duration. Irrigation of the bowel caused a disappearance of the abdominal tumour; but as the symptoms continued, the abdomen was opened and the intussusception was found to have been completely reduced. The child was free from any bad symptoms for two days, but at seven p.m. on the second day the intussusception suddenly returned, and a well-defined tumour could be felt within the abdomen. This tumour again disappeared after the bowel had been filled with salt solution, but the symptoms again recurred at 10 p.m., and the child died the same night. At the post-mortem examination a large ileo-cæcal intussusception was found with its apex reaching to the brim of the pelvis on the left side. The duodenum was so much constricted by the tense edge of the mesentery as to cause partial obstruction of its lumen. The intussusception was easily reduced by the introduction of two pints of water into the large intestine under a moderate pressure.

These cases show, I think, the danger of treating recurrent intussusceptions by irrigation or inflation, and it is impossible to tell beforehand whether or no an intussusception will recur. It was quite impossible in case iii for irrigation to reduce the small piece of invaginated bowel, which was stiffened by inflammation, especially as the sacculi were bound together by the products of a plastic exudation. Yet, so long as the bowel remained invaginated, even to the slightest extent, so long did the intussusception show a tendency to recur. Abdominal section therefore was the proper and only effectual method of treatment, for when perfect reduction had been made there was no further recurrence. Irrigation may be used to mitigate the sufferings of the patient until the services of a surgeon accustomed to abdominal work can be obtained; or, it may be used by the surgeon himself to reduce the size of the tumour immediately before he operates. I am confident, however, that this method of irrigation should not be used to the exclusion of abdominal section, and medical men should urge parents to allow an operation to be done at the earliest possible opportunity in every case of intussusception.

With the rarest exceptions recovery from an intussusception only takes place when the invagination is capable of easy and simple reduction. Fifteen of the sixty-five cases in my table needed excision of a part of the bowel before the intestinal obstruction could be overcome. Fourteen of these cases died, and one recovered. In ten cases excision was performed because the bowel was gangrenous; in four cases because the softened tissues tore during the manipulation required to reduce the invagination; whilst the notes in the fifteenth case only say that death followed the excision of the bowel for the relief of an intussusception. In six of the fifteen cases an artificial anus was made, in five cases a Murphy's button was employed, twice Maunsell's operation was performed, and once the intestine was united by a circular enterorrhaphy. The case which recovered was operated upon by my colleague, Mr. T. Pickering Pick. The opera-

Digitized by Google

tion was performed on Good Friday, 1896, in a girl æt. eight and three quarter years who had suffered from symptoms of intussusception for six and a half days. On opening the abdomen the invagination was found to be of the ileo-ileac variety, and the bowel gave way during the attempt to reduce the intussusception. Mr. Pick immediately cut away the injured part and united the ends by a Murphy's button. She made a good recovery and passed the button on the ninth day. Mr. Pick published an account of the case in 'The Quarterly Medical Journal,' Sheffield, vol. v, pp. 121-123.

I saw this patient on Monday—July 22nd. She seemed to be in perfect health, and is just beginning to menstruate. She has no indigestion, but her mother says that she is "faddy" in her appetite. She never has any abdominal pain, and her bowels are open regularly. There is a gap between the recti abdominis muscles, but there is at present no tendency to hernia.

"Nineteen Years' Experience with Creosote in Tuberculosis" was the title of a paper read by Dr. A. Burrows at the fifty-second meeting of the American Medical Association. The author gave no statistics, but asserted his conviction from personal observation that creosote in larger doses has a curative action in pulmonary tuberculosis. The ordinary dose of five or ten minims is too small, except to benefit a feeble digestion. It is only in large doses (60 to 100 minims) three times a day, by the mouth (given in cod liver oil, whisky, or cream), supplemented by the inhalation of fifteen to twenty minims daily (in some hydro-carbon oil), that the curative effect upon the lungs is obtained. He commences with a dose of twenty, and rapidly runs it up to eighty, minims of beech-wood creosote, given after a full meal, three times a day. He had not observed any cases of renal disturbance or other bad effects from these large doses, but had seen some remarkable cases of recovery.—*Philadelphia Medical Journal*, June 22.

TREATMENT OF CANCER OF THE TONGUE.

MR. BLAND-SUTTON.

"The excision of the anterior portion of the tongue, or the right or left anterior fourth of the organ when the disease is localised to one side, is an operation devoid of risk or difficulty. When the disease deeply invades the tongue, involves the floor of the mouth, or extends so far backwards that, in order to get beyond the limits of the disease, the surgeon interferes with the pillar of the fauces, then the operation is often hazardous. The chief difficulty is connected with hæmorrhage, and in order to obviate it a variety of methods have been advocated for the excision of the tongue. A careful selection of cases, wide removal of the diseased tissues, and removal of the infected submaxillary lymph glands are the points to bear in mind. It is an important point in operating upon the tongue to avoid the entrance of blood into the trachea, as it is then drawn, during inspiration into the lungs, and gives rise to septic pneumonia. Should blood in considerable quantity get into the trachea it may cause suffocation. To avoid these complications it is useful, in extensive operations on the tongue, to perform laryngotomy and administer the anæsthetic through a laryngotomy tube, and in order to prevent blood from getting into the trachea, the pharynx is plugged with a sponge. The mortality of operations for the removal of epitheliomatous tongues is not less than 10 per cent.; the chief causes of death are hæmorrhage, septic pneumonia, and asthenia. Although after excision of an epithelioma of the tongue recurrence in the stump or cervical lymph glands within a year of the operation is the rule, nevertheless it is in some cases delayed for five and even seven years. It is also useful to bear in mind that, in some cases, where the disease is advanced and too extensive to admit of removal, the pain may be relieved by division of the lingual nerve, and a few patients are rendered comfortable by ligature of the lingual and facial arteries."

We have abstracted the foregoing passage from the new edition of Mr. Bland-Sutton's valuable work on "Tumours—Innocent and Malignant," recently issued by Messrs. Cassell and Co. The new edition contains many emendations and additions, and is illustrated by over three hundred engravings.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 460. WEDNESDAY, AUGUST 21, 1901. Vol. XVIII. No. 18.

CONTENTS.

PAGE

*A Clinical Lecture on Tubercular Peritonitis. By W. HALE WHITE, M.D., F.R.C.P. 273

*A Surgical Consultation at the Medical Graduates' College and Polyclinic. By JAMES BERRY, F.R.C.S. ... 278

*A Pathological Demonstration at the Hospital for Sick Children. By J. H. THURSFIELD, M.D. 284

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A CLINICAL LECTURE
ON
TUBERCULAR PERITONITIS.

Delivered at Guy's Hospital, June 5th, 1901.

By W. HALE WHITE, M.D., F.R.C.P.

GENTLEMEN,—Before going over the case which will form the subject of to-day's lecture, perhaps I had better tell you exactly what tubercular peritonitis is. It is a peritonitis set up by the tubercle bacillus, and can be easily produced in animals by injecting tubercular sputum into the peritoneal cavity. It is very rarely pure infection; before long other micro-organisms are associated with the tubercle bacillus.

Clinically it appears to be much more common in women than in men, but whether that is so is doubtful, for post-mortem experience appears to show that the two sexes are affected almost equally. The matter, however, is of small importance, as tubercular peritonitis occurs frequently in both sexes. The frequency of the disease appears to vary in different parts of the world, for different authors give the percentage of deaths due to tubercular peritonitis as varying between 1¼ and 16 per cent. The higher number was obtained by Borschke, who, in 2450 autopsies, found 1393 cases of tubercle, and of these 226 or 16 per cent. had the peritoneum affected. Most morbid anatomists will tell you that primary infection of the peritoneal cavity by the tubercle bacillus is very rare. Wilks and Moxon doubt, however, whether it is as rare as most people think. Be this as it may, all are agreed that a frequent source of infection is tubercle of the female genital organs. The disease varies very much in its frequency at different ages. Bottomley gives the following:

Under	10	years of age	.	.	30 cases.
10 to	20	,,	,,	. .	112 ,,
20 to	30	,,	,,	. .	137 ,,
30 to	40	,,	,,	. .	122 ,,
40 to	50	,,	,,	. .	93 ,,
50 to	60	,,	,,	. .	31 ,,
60 to	70	,,	,,	. .	11 ,,
Over	70	,,	,,	. .	2 ,,
					538 ,,

Digitized by Google

Before you can understand the clinical symptoms you must have a good mental picture of the post-mortem condition. It is remarkable that tubercular peritonitis is so rare in comparison to the frequency with which sub-peritoneal tubercles are found underneath tubercular ulcers of the intestine. In the simplest cases of tubercular peritonitis you will see a number of grey tubercles, none of them larger than swan shot, scattered over the peritoneum. They are found mostly under the diaphragm, and are rarest over the intestine. In such a case as this you will often find much fluid in the peritoneal cavity. In the most common form of tubercular peritonitis the striking thing is the enormous mass of adhesions; all the abdominal organs may be got out of the abdomen as a solid whole, as though they had all been fixed by having plaster of Paris run into the abdominal cavity, but the abstraction of them is by no means easy, for there are many adhesions to the abdominal wall itself. I have in such a case spent a couple of hours trying to dissect out the intestine from the matted mass, and then given up the task as hopeless. When a cut is made across this mass, a number of sections of the gut are seen, and they do not collapse when they are empty, for they are held open by adhesions. The intestinal walls are much softer than usual, so that they are torn in trying to get the intestines out. Among these adhesions there are numerous cavities containing fluid which may be purulent, and these abscesses commonly discharge themselves into one or more adjacent pieces of intestine, so that all sorts of fistulous communications are formed, and sometimes such a collection discharges externally on the one hand, and into the bowel on the other, so that there is a fæcal fistula open externally. In cases in which the adhesions are less marked, although extensive, the omentum is commonly puckered up and thickened, so as to form a transverse tumour lying across the abdomen just below the transverse colon, and various other masses of the thickened peritoneum occur in different parts of the abdomen. The peritonitis may be entirely fibrous, but caseous masses are commonly present, and the mesentery is often so puckered that the intestines are drawn back to the spine. Usually not much fluid is present in these cases, and it is commonly loculated. It varies in consistency, but is usually turbid and not rarely purulent; it may contain blood. The abdominal lymphoid glands are usually caseous, but you must not suppose that the enlarged caseous mesentery glands are often felt during life. When masses supposed to be these glands are felt, it nearly always turns out that they are really hard masses of peritoneum affected by tuberculous disease. You will find cirrhosis of the liver frequently associated with tubercular peritonitis.

Now I will give you the particulars of the patient that forms the subject of this clinical lecture. He is a young man aged nineteen, who came into the hospital on April 24th, stating that for about a fortnight he had been out of sorts suffering from abdominal pain, and lately felt so ill that he had not been able to do his work. His bowels had been irregular. On admission his temperature was 103°; he complained of no pain except in the abdomen, especially in the neighbourhood of the umbilicus; his breath was very foul; his tongue was covered with a light brownish fur; the abdomen was tumid and distended, it moved with respiration; no spots were seen. On palpation it seemed abnormally resistant, the spleen could not be felt, the front of the abdomen was resonant all over, there was tenderness on percussion over the umbilicus, and there was dulness in both flanks, which disappeared when the patient turned over. In the right axillary region there was an impaired note on percussion. No Widal reaction could be obtained, and the report on May 4th says that the abdomen was gradually getting more distended; there was more fluid in it. No typhoid spots had been seen. There was some dulness at the apex of the left lung. On May 9th, as the abdomen was still distended, the patient was seen by Mr. Dunn with a view to operation. It was also discovered that he had fluid at the base of his left lung; this was aspirated, three pints being drawn off. On May 11th a pleuritic rub was heard on the right side of the chest. On May 14th Mr. Dunn opened the abdomen just below the umbilicus. The peritoneum was found to be very thick, and when opened a quantity of serous yellowish fluid gushed out. The intestines were matted together, and tied down to the posterior abdominal wall. The abdominal cavity was flushed out with warm sterilised water, and the wound was then sewn up. For about a week after the operation the patient seemed a

Digitized by Google

little better, but then the temperature began to mount up again, and from then till now it has ranged about 101°, and the signs of tubercle in the chest have increased considerably.

I will now tell you something more about the symptoms. In the first place this man illustrates that the disease may be met with in adults, though it is a common impression that it is chiefly found in children. He also illustrates that, although it is not often primary in the abdomen, it is first noticed there. The early symptoms are that the patient becomes out of health and languid, his appetite is poor, he suffers from indigestion, irregular bowels, sometimes constipation, sometimes diarrhœa. He complains of abdominal pain, the tongue is furred, and he notices that the abdomen is distended. It is then discovered that his pulse is more rapid, his temperature is up a little, and he may sweat at night. Gradually all the above symptoms become worse, the abdomen more distended and particularly tympanitic; the reason for this being that the bowel is easily paralysed in this disease, gas collects in it, and makes the abdomen tympanitic and distended. If fluid is present, as it was in our patient, there is dulness in the flanks, and if the fluid is not shut in by adhesions this dulness shifts when the patient is shifted. By now the thickened omentum can be often felt transversely across the abdomen, the abdomen is lumpy, the umbilicus prominent and red, the patient anæmic, he loses weight, and his pulse becomes more rapid and his temperature higher. Later on, as our patient shows, all the above symptoms become worse; he becomes very wasted, the skin is shrunken, dry, and brown, and there are few things more striking in medicine than the extreme distension of the abdomen in marked contrast to the general wasting. The anæmia becomes profound, the feet swell, and slowly the patient sinks.

The only details that I need call your attention to are in connection with the abdomen. The condition of this will obviously be very variable in accordance as to whether it is distended by fluid, gas, or both. Adhesions, too, must make the state of the abdomen different in different cases. You must never forget also that loculi form, and they make the detection of fluctuation very difficult. You can sometimes press the fluid from one loculus to another, when you will get a gurgling sound. Another thing to remember is that you may find the fluid sometimes as much in front of the abdomen as at the sides. This is because the mesentery shrinks and drags back the intestine. Then, too, because the amount of gas in the intestine varies very much from day to day, the ease with which you will feel the omentum and the lumps of tuberculous matter will vary as well. Then, too, never forget you may get a fæcal fistula, and if this occurs it is always, or almost always, at the umbilicus; but apart from a fæcal fistula, the umbilicus is often very prominent, and it may be red. Peritonitis, too, may make micturition difficult. With these patients the appetite is often voracious, and they have most extraordinary likings for out-of-the-way articles of food; they suffer much from constipation and diarrhœa, and their motions are foul.

Some authors have classified the cases into acute and chronic, and have subdivided the chronic into fibro-caseous, ascitic, and fibrous.

It is, however, very important for you to remember that the groups merge much into each other, and further, as time goes on, a case that at one period of its history would have been placed in one group passes into another.

With regard to the complications we have no time to say anything about tubercle in other organs, and we must therefore restrict ourselves to the abdominal complications. Of these by far the most important is a fæcal fistula, and I have already explained to you how this is formed, and that it occurs usually at the umbilicus; it is more frequently seen in children than in adults. The second complication is that a fistula may form between adjacent parts of the bowel in much the same way as a fæcal fistula forms externally, but fistulous communications between adjacent parts of the bowel are only diagnosed when the communication is between the jejunum and the colon. Then as the food short-circuits, and so does not pass through the greater part of the intestine, the patient wastes very rapidly, and much undigested food is found in the motions.

The third complication for you to bear in mind is intestinal obstruction. This may be due to a band, but this is rare; by far the most frequent form of intestinal obstruction following tubercular peritonitis is that which was first described in the 'Guy's Hospital Reports' by Dr. Fagge as obstruction due to compression and traction. These

Digitized by Google

words exactly tell you what happens, for owing to the contraction of the tubercular peritoneum it compresses the gut against the back of the abdomen, and at the same time the shrinking mesentery draws it down to the back also. Very slowly and very gradually therefore intestinal obstruction supervenes in these cases, and naturally it forms one of the most difficult varieties of obstruction to treat.

The diagnosis of tubercular peritonitis divides itself into two stages; in the early stages it is difficult to diagnose from any slow wasting disease in which the patient gradually loses health, but it is very important to remember that abdominal pain usually puts us on the right track, and whenever you have before you a patient at the age at which tubercular peritonitis is most common, and who is slowly getting out of health and losing flesh, and complaining of abdominal pain, you must always bear in mind the possibility of tubercular peritonitis.

The other condition from which it is difficult to diagnose it in its early stages is typhoid fever, and that difficulty presented itself in the patient before us, for he did not give a long history, and he was seriously ill when he came in; but, as in him, the disease soon declares itself one way or the other, for you either detect signs of peritonitis, as we did in his case, viz. the fluid in the abdomen, or, on the other hand, you get the well-known symptoms of typhoid fever. Occasionally, but very rarely, you may tell the two diseases by observing that the temperature is higher in the morning than in the evening, this pointing to tubercular peritonitis. In the latter stages the difficulties of diagnosis always takes the shape of determining what is the cause of ascites. That we cannot go into to-day, but I would remind you that if you decide the ascites is encysted, then it is almost certainly tubercular. Further, feeling the thickened omentum passing transversely across the abdomen is very strong evidence that the case is one of tubercular peritonitis.

The prognosis is unfortunately bad, and the points to which you must direct your attention in estimating it are, first of all, you must find out if there is tubercle in other organs, and this may quickly determine the prognosis, as, for example, if there is tubercular meningitis.

Severe gastro-intestinal symptoms may too quickly determine the prognosis, for it is obvious that if the patient is suffering from severe vomiting and diarrhœa which you cannot check, he will soon go down hill. Then, too, severe toxæmic symptoms, such as profound anæmia, great wasting, high temperature, rapid pulse, and albuminuria are all bad. Whatever the cause may be, loss of weight is extremely bad.

Again, these patients always do badly if they have been taking too much alcohol, or if they are suffering from active syphilis. The cases in which there is a fæcal fistula never do well, and speaking generally the prognosis is much better just in proportion as there is a large quantity of ascitic fluid present in the abdomen.

The treatment naturally divides itself into general and local. The general treatment is such as you would apply for tuberculous disease in any part of the body. The patients should have abundant fresh air day and night. You should send them into the country, where they can lie out of doors all day, and have their windows open all night. You should feed them up with plenty of good food, and clinical experience and experimental work both show that these tubercular patients do better upon plenty of farinaceous and fatty food than upon proteids. In England it is customary to apply some mercurial ointment to the abdomen, and that is best done by taking equal parts of lanoline and the pharmacopœial ointment of the oleate of mercury, smearing them on a flannel binder, and fastening this round the abdomen. I myself think that this does some good, but how I do not know. In France it is customary to apply iodine locally.

Passing now to surgical treatment, all authorities are agreed that laparotomy is much more reliable than puncture. After the abdomen has been opened some surgeons wash it out with boiled water, others use weak antiseptics, others dust iodoform on some parts, others excise caseous nodules, and some take away the Fallopian tubes if the genital organs are tuberculous; but as all surgeons appear to get equally good results, it seems clear that the chief point is merely opening the abdomen; but why this should be so no one has satisfactorily explained. The points for you to beware of particularly are that the intestines are very soft, so that you may easily tear them: the adhesions may be very numerous, or it is difficult to get into the peritoneal cavity at all, and you

Digitized by Google

must be very particular not to do anything that may lead to the formation of a fæcal fistula. Experience has shown that it is better after the operation to sew up the abdomen rather than to put in a drainage tube, and probably it is wise not to take away the tubercular female genital organs unless their removal is particularly easy. The mortality from the operation alone is from 2½ to 3 per cent.

Now you will want to have some guides as to whether you should operate in any particular case. In the first place, never operate if the tubercular peritonitis is merely part of an acute general tuberculosis. In the next place, never operate if there is already a fæcal fistula, for the presence of a fæcal fistula shows that there must be numerous adhesions, and you will in all probability not be able to get into the peritoneal cavity at all. Next remember that you will get your best results in the cases in which there is most fluid present. It is quite true that those are the cases which will do best whether you operate or not, but on the other hand, they are just the cases in which the risk of operation is exceedingly small, and therefore, in them, it is probably wise to open the abdomen and let the fluid out. Certainly you should do so if sending the patient away into the fresh air is impossible, but probably the best thing would be to open the abdomen, let out the fluid, and then send the patient away into the country. The worst results are obtained when there is little or no fluid in the abdomen. Therefore, in these cases, you certainly should not open the abdominal cavity. Localised collections of fluid should be dealt with on the same principle, but you should be very wary of touching them because the very fact that the fluid is localised shows that there are many adhesions, and shows it is not the class of case that will probably do well. Never incise the prominent umbilicus that is so often met with in these cases, for it very often means that there is a tubercular abscess under it, and sooner or later this bursts into the bowel, and if you have previously opened it you have a fæcal fistula left. Another important point is that if there is already a fæcal fistula formed, do not on any account try to deal with it surgically. I have seen the most disastrous results follow, and I have never seen any good done. In talking to the friends about the operation it is only fair to them to say that while the immediate effect of an operation is often good, nevertheless the symptoms not infrequently return. You will remember the patient we have been discussing today was for some time better, but now I doubt if he has been much improved by the operation; nevertheless, post-mortem experience does show that there may be a complete cure. Lastly, it is interesting to note that operation for tubercular peritonitis sometimes benefits tubercle elsewhere in the body. Cases have been recorded in which distinct improvement has taken place in pulmonary tuberculosis after operation for tubercular peritonitis.

Reuter has examined the bodies of twenty-two cases of strangulation, seven of them suicides. Six others were adults, nine infants. He describes the anatomic conditions found, believing that cyanosis of the face, with or without ecchymoses, and hæmorrhage of the conjunctiva and eyelids are noted in every case externally; internally are seen congestion of the brain and meninges, hæmorrhage into the soft parts of the neck, near the line of strangulation, or at some distance above it, and marked injection of the mucous membrane of the upper air-passages. In infants, while the external conditions are found the same, only congestion of the brain and its coverings is seen internally. The mechanism of death by strangulation or by hanging is the same, yet the lesions found are different. Reuter has looked over the statistics of 300 cases of autopsy after hanging. He states that there are, as a rule, no cyanosis of the face, no marked congestion of the brain or meninges, and no hæmorrhage into the muscles of the neck; that injuries to the larynx are frequent; and that rupture of the intima of the carotids is frequently found. Reuter concludes, after quoting a few cases, that the differential diagnosis of death from strangulation or hanging, should not be difficult when the anatomic conditions found have been thoroughly examined.—*Philadelphia Medical Journal*, July 13th.

Digitized by Google

A SURGICAL CONSULTATION

At the Medical Graduates' College and Polyclinic, June 12th, 1901.

By JAMES BERRY, F.R.C.S.

I HAVE several cases to show you this afternoon, so I shall not be able to give very much time to any one of them.

Jaundice due to Cancer of the Common Bile-duct.

I will take first of all the case of this woman who has been sent up by Dr. Wylie. These are the notes which he has sent with her: "She is fifty-six years of age, and she has not felt well for the last twelve months. About three months ago she had some pain over the liver, followed by jaundice and loss of flesh. On examination the liver was enlarged, and the gall bladder was felt extending to the right iliac region, and of about the size of a small soda-water bottle. It is about one third the size now. There is no history of cancer in the family." Those notes give you all the main features of the case. On looking at her we see at once that she is deeply jaundiced; she is not merely yellow, but she has got the brown tinge which one sees so often in advanced jaundice. The yellowness affects the conjunctiva and all parts of the body. Notice the deep yellowness of the whole of the abdomen. When she sits up you will notice a certain amount of separation of the recti, allowing the abdominal viscera to bulge. That has nothing to do with the rest of the trouble. We may also notice that she has several small red spots about the body, evidently the result of scratching, and that leads one to ask whether the skin does not itch a good deal. Itching is a very common symptom—indeed one of the most distressing symptoms of deep jaundice. This is simply part of the jaundice. With this amount of deep jaundice at this age it is not very likely that it will be anything except one disease which we will mention presently after the patient has left us. But it is possible there may be other causes for it. I naturally refer to carcinoma, involving the bile-ducts somewhere; whether in the liver, in the pancreas, or between the two organs I do not yet know. On examining the abdomen I see it is moderately full and soft, and directly I lay my hands upon it I find in the right hypochondrium a round, fairly movable swelling, which feels like a large egg, quite smooth, immediately below and to the left of a distinct edge running obliquely across the abdomen, which is obviously the edge of the liver. Above this edge I feel a smooth convex surface, extending right up to the ribs on the right side, and which is evidently the liver. On percussing this I find it is dull all over. There is no intestine in front of the swelling. On placing my hands in front of and behind the swelling I can feel this liver very plainly, both from behind and also in front; there can be no question at all that this is a large liver, and there can be no doubt that the egg-shaped swelling is an enlarged, tense, gall-bladder. I cannot feel anything in the nature of a lump on the surface of the liver; so far as I can tell it is quite smooth. There is no evidence of any secondary deposit in the liver; though of course I do not say there is no secondary deposit. I will now ask the patient a few questions about her symptoms. She has told us about the itching, and we know about the jaundice. She takes her food very well, she has not been sick; the bowels are quite regular, but the motions are white. She says her water is very brown, almost like beer. She experiences no pain whatever. She had pain, but that was before the jaundice came on. She says she is sure she has been losing flesh. You will notice I do not ask her a leading question, but asked her whether she knew what her weight was, and she at once said she was losing flesh. I have no doubt that is correct. I do not think there is any fluid in the abdomen.

With regard to the diagnosis, we have evidence of complete obstruction of the bile-duct. We know she is an elderly woman. The jaundice is progressive, and it is now deep; it has never gone away and then come back again. Now, progressive jaundice in an elderly person is almost certain to be due to carcinoma. Whether it is carcinoma of the pancreas or of the pylorus, or of the liver itself, is a point which may be discussed now. A large and tense gall-bladder shows that the growth is in or below the cystic duct. If the carcinoma were in the liver above the cystic duct, involving the bile-ducts in the liver itself, we should not expect to get such a tense distended gall-bladder as that. If the growth were in the cystic duct—not a very common place—we should get an enlargement of the gall-bladder, but we should not get such a very large liver. So the evidence shows

Digitized by Google

that the cause of the obstruction is in the common duct, or near it. The most likely place, of course, is at the head of the pancreas. It is possible that there is a growth somewhere outside the pancreas, pressing upon and involving the duct just before it reaches the pancreas. I have mentioned carcinoma of the pylorus, but I put that on one side because she has no vomiting, and no evidence of any obstruction of the pylorus; she has not vomited blood, and she takes her food well. I have no reasonable doubt that it is a case of carcinoma of the pancreas. A question which ought to be discussed perhaps is, can the obstruction be due to gall-stones? The pain she had at the beginning might possibly point to gall-stones, but it is a very unusual thing for gall-stones to cause such intense jaundice, that is to say, progressive jaundice, without a history of gall-stone pain or of attacks of jaundice for a very long time before. I have never yet seen a case of deep jaundice in a person over fifty that had steadily progressed, which had never been preceded by another attack or by gall-stone colic which did not in the end prove to be carcinoma. I think there is no reasonable doubt that she has carcinoma of the pancreas, possibly carcinoma of the common bile-duct.

With regard to treatment, of course removal of the growth is out of the question. The only point of practical importance is whether it is worth while to relieve her jaundice. That, of course, could be done in one of two ways: (1) by opening the gall-bladder and giving her a fistulous opening. That would be a miserable condition for her, because she would then have her bile running away, and possibly might be no better off than she is now; (2) one might establish an artificial fistula between the gall-bladder and some part of the intestine, such as the duodenum. It is open to question whether her trouble or distress are sufficiently great to make it worth while for her to undergo the annoyance and the risk which an operation of that kind involves. The itching of the skin seems to be the chief trouble, and sometimes in these cases it is so intolerable that it is worth while to operate for this reason alone. I should recommend that the matter be put to her in that way—that if her distress is great she can be relieved by an operation, and on the whole it is to be recommended. Of the two operations I think cholecystenterostomy is better than the formation of a fistula. But it is well to bear in mind that these operations upon patients who are deeply jaundiced are dangerous, far more so than are the operations for gall-stones in the cystic duct. The ordinary case of large gall-bladder due to gall-stones in the cystic duct is one in which one operates very readily indeed, because the mortality is very small and the relief very great. Cases which are deeply jaundiced one does not operate upon at all readily, because one knows that these people are very apt to bleed seriously. In them even a comparatively small operation is not free from risk of very serious, and even fatal hæmorrhage.

Fracture of Fore-arm, with paralysis of Ulnar Nerve.

The next patient is a girl twelve years of age, and she is the first of several cases of injury which I have to show you to-day. This case was sent to us by my colleague, Mr. Roughton, who, however, only saw the case for the first time two days ago. She comes complaining of deformity of the hand, and it is obvious that the deformity is very considerable. Some seven weeks ago she fell on her wrist and broke it, and it was put up in splints. It is obvious from the shape of the hand that there is either fracture or dislocation, or separation of the lower epiphyses of the radius and ulna. On looking at the ulna I can see it is not quite straight, that there is fracture of the lower end of the ulna, possibly with separation of the epiphysis. Then, too, we notice that the lower ends of the radius and ulna are carried somewhat backwards. The styloid processes have their normal relation to the carpus, therefore there is no dislocation, and the lower ends of the radius and ulna have been carried back together. There is either fracture through those two bones, or possibly separation of the epiphyses. It is a very difficult point to determine at this stage of the case which of the two it is, unless one can get a skiagram of the part. Mr. Roughton has recommended that it should be skiagraphed, and that, of course, will give us more information about the nature of it.

With regard to union, it is quite obvious that the fracture has united. On putting my right thumb on the head of the radius and rotating, I find that the radius moves as a whole, and the ulna is obviously united. The bones have united

Digitized by Google

in the position which you see here. But there is something more. Those fingers are peculiar in their shape and the way in which she holds them; she puts fingers and thumb together very incompletely. She has no power of separating the fingers. The first row of phalanges tend to be extended, and the next two rows of phalanges tend to be flexed. There is a tendency to extension or hyper-extension of the first row of phalanges, and flexion of the others; in other words, the interossei do not act. There are also other evidences of ulnar paralysis; the hypothenar eminence is obviously wasted, and the inner part of the thenar eminence is also atrophied; the outer part of the thenar is probably wasted owing to disuse. Although the ulnar muscles are paralysed, the others are not. I will not delay to test her sensation now, but I have been told she is anæsthætic on both sides of the little finger. The next thing to consider in any case of nerve injury is the question of trophic changes. Have any changes taken place in the skin, hair, or nails? They are not very marked in this case, possibly because the paralysis has only been in existence for a short time. One would like to examine with the battery to see whether the muscles are completely paralysed, and whether there is the reaction of degeneration. I have not time or opportunity to do so here, and I do not know whether Mr. Roughton has done so yet. The information obtained from the battery would help us to determine whether the ulnar nerve, which is obviously affected, is completely divided, or merely involved in the callus. There is not much doubt that it is due to involvement in the callus, and not due to section of the nerve, though it is conceivable that in the injury the nerve might have been cut across. Still I think the latter supposition is so unlikely that it is not worth consideration.

Then comes the question of treatment. Is the deformity worth treating? I see no objection to the examination of the limb under an anæsthetic, but I should not expect that very much good would accrue from any attempt to restore the shape of the bones seven weeks after the injury. The bones are clearly well united. There is not enough deformity in the bone to make it desirable to do anything in the way of refracturing it. With regard to the nerve, I think it is probable that Mr. Roughton may see his way to doing something to free the nerve from the callus. That is a point I should leave to be investigated after a more thorough examination of the parts, perhaps under an anæsthetic, certainly with the battery. I believe she will have a very useful hand, though of course not a very shapely one.

I had hoped to have been able to show you a very good companion case to this—that of a woman who a year ago fell and broke her wrist. She went to a hospital—not mine—and the arm was put up in splints, and for some reason or other, which I do not understand, sloughing of the skin took place and the forearm suppurated very badly; she had a very bad abscess on the forearm and a large abscess on the back of the hand, and when the splints were taken off, ten days after the injury, there were very extensive sloughs. She says that the house surgeon would not take the splints off when she complained of pain; but if the house surgeon were here we should possibly find that she was told to come and show herself the day after the injury, but failed to do so. However, the splints were left on, and there was undue pressure and very extensive cellulitis. She was taken into the hospital, and came out of it with her hand terribly crippled. She had extreme atrophy of the lower half of the forearm; there were the marks of the incisions where the abscess had been opened, and a very typical hand showing median nerve paralysis. Upon cutting down upon the median nerve, one and a half inches of it were found to be firmly bound down by adhesions, the result of this cellulitis. The nerve was freed, and she is now slowly regaining the use of her hand.

Old Fracture near the Ankle.

Here are two or three cases of injury to the lower limb. This man was sent up by Dr. Handcock. It is very obvious there is some swelling about the ankle. He is forty-one years of age, and he was hurt in March of last year. The notes I have with the case state that the left leg was broken and the fibula driven through the skin: there was displacement of one of the bones of the joint, which was greatly contused. The whole of the tendons surrounding it were contused, and there were numerous ulcers in the foot due to pressure of splints, wasting of muscles, and so on. That is the condition when the patient was sent to Dr. Handcock in October last year. The question

Digitized by Google

is whether the limb is likely to be of any service, and what the treatment should be. He complains of much pain from the knee downwards, and that pain is worse when he is walking. It hurts him when he stands. It is very obvious there is a swelling above or about the ankle. It is evidently tender. On comparing it with the one on the other side, it is obvious that he must have had a fracture about the lower end of the tibia and fibula, and of what nature or kind one does not know exactly. But there is very considerable thickening of the bones at their lower ends. The foot was turned outwards. There is no œdema and no evidence of suppuration. I ask him about a tender spot, because I have in my mind the possibility of his having a small piece of necrosed bone and abscess in the tibia. But there is no one spot there which is extra tender. He says the pain is worse in the daytime, when he is on the leg. There is not much movement, but he says there is a good deal more than there was. The foot and the leg form a right angle, but I cannot bring the foot within the right angle. I ask him to try to walk on it, but he cannot do so with comfort. The foot as a whole is situated just below the axis of the leg; it is not one of those bad cases where the foot lies well to the outer side or to the inner side of the axis of the leg. This has had very good treatment as far as the position is concerned. Since he has had the battery to the muscles it has been getting better. He has practically had all the best treatment for it—battery, rubbing of the muscles, iodide of potassium, and so on. There is no heat about the part, nor any evidence of the joint being affected by suppuration; but there is chronic periostitis. I should advise him to go on with that treatment as it is getting better, and if the pain continues very long, or if it gets worse, I think the question should be raised whether it would not be worth while to have an incision made down upon this bone and have some trephining operation done, which very often relieves chronic pain in these cases. I think the possibility of there being a small amount of necrosed bone, or perhaps an abscess there, should be borne in mind. It is a very common type of case, but one often sees even more deformity after Pott's fracture. In such cases one sometimes has to do an open operation for the restoration of the foot to its normal position.

Old Injury to the Ankle.

This next case is that of a man who caught his foot in a machine some months ago, and was laid up for a considerable time. This is a very similar case to the last one, but not such a marked one. I think there is some transverse widening between the malleoli. The movements are much better than in the last case. There is little pain in the joint. If the pain continues or becomes worse or very severe one might do the same thing that I mentioned in connection with the other case; but I do not think it will be necessary. I think with rubbing and electricity the condition of the muscles and ankle will get better in time. You will notice that the foot is at a right angle, and that it is in its correct position with regard to the axis of the leg.

Chronic Ulcer of the Leg.

Here is a man who had an injury to the foot twenty-three years ago, owing to a piece of timber falling on it. It is not a fracture. For some years he has had an ulcer there, which we hear heals up every now and then. It is the ordinary story. When the patient rests and does not walk about the ulcer heals up, but when he gets about on it it breaks down again. On previous occasions we hear it has always healed up. The last time it was bad was five years ago. Then it began to get bad again two months ago, and since then it has not healed up. You see a very small characteristic chronic ulcer on the malleolus, the skin around which is discoloured with old chronic inflammation. There are varicose veins also. With regard to the ulcer itself it has a well-defined edge, no eversion, and the edge is not harder than the surrounding skin. The base of the ulcer is very indolent. It is a typical indolent chronic ulcer. The movements of the foot and ankle are fairly good, and there is no evidence or history of syphilis. No doubt the ulcer was started originally by the injury, and is kept up now by the bad circulation in the skin as shown by the varicosity of the veins. As the ulcer has healed up on previous occasions by rest I should hope that rest, together with most scrupulous cleanliness, washing it freely with weak perchloride of mercury solution, will cause it to heal up. If it does not do so I should be inclined to recommend the practice, which I think is now carried on more generally than formerly, namely, cutting the ulcer right out. Several times lately I

Digitized by Google

have dealt with a very chronic ulcer in that way. The ulcer and the neighbouring hard unhealthy tissue is cut out, and then skin, cut by Thiersch's method from some other part, is grafted upon the wound. If you can get a really clean wound that skin is likely to grow, and then the patient has a good piece of skin in a place which formerly was occupied by unhealthy skin. There is, of course, always some possibility that you might not keep the part perfectly clean and aseptic, and then some part of the skin put on may slough. If the patient is in good condition, and the surrounding skin is not very unhealthy, if the ulcer is sufficiently intractable and has not yielded to ordinary methods of treatment, the practice of excising a chronic ulcer is not to be overlooked. Sometimes I have dealt with even large ulcers in that way, with the help of Thiersch's grafting.

Spinal Caries.

This little girl was brought to me at the Alexandra Hospital for Hip disease. The complaint made was of weakness and pain in the leg. On asking the mother whether she complained of anything else, she said, "Yes, she complained of a good deal of stomach-ache, and was constantly crying and putting her hand on to the stomach." A very brief examination made one say it was not a case of hip disease, and therefore it was not suitable for the Alexandra Hospital, but I thought it would be a good case to show you, so I sent her on here. The legs are not wasted, there is absolutely free movement of both hip-joints; therefore there is no hip disease. With regard to the pain in the abdomen the abdomen is rather full, but there is nothing obvious to be felt. On questioning the mother about the pain she told me there was some pain in both thighs. There is therefore pain in the stomach and pain in both thighs and weakness in the legs in a young child, so one immediately thinks of spinal caries, and we at once look at the back. There you can see a very well-marked angular curvature in the mid-dorsal region, forming a very sharp angle. I ask her to pick up my keys from the floor, but she does not do it in the hesitating way in which she would if she had acute disease. She just rests her hand on the thigh on getting up, but that is all, which means that the condition is not acute. I lift her up with my hands in the axillæ, but she does not complain of pain. On the other hand, the disease is not quite cured; if it were, she would not have pain in the stomach. The deformity, then, is a definite angular curvature—in this case conclusive evidence of caries of the spine. Having diagnosed that condition, there are two things we must always think of. One is, is there an abscess? I have examined with my fingers in the iliac fossa of both sides, but I can find no abscess. Mind, I do not say there is no abscess; it is not at all unlikely that there is a small abscess deep down. The other inquiry is, Are there any nervous symptoms? Is there any evidence of suppuration behind the bodies of the vertebræ, pressing on the cord? Or, is there evidence of the bodies themselves pressing on the cord? The original complaint was weakness of the legs. It is obvious that the reflexes are increased, and that is indicative of slight pressure. I have very little doubt that if this child were kept quiet the exaggeration of reflexes would disappear. It is a chronic quiet case, and the prognosis is fairly good if the child be kept quiet. I have recommended that she be taken to a general hospital and have a poro-plastic apparatus made for the back. She should be kept quiet for months, and have a careful watch kept for the appearance of an abscess.

Scirrhus of the Male Breast.

This man is a patient of Mr. Roughton's, sixty-two years of age, and I should like to express my indebtedness to Mr. Roughton for kindly allowing me to show his cases. Nearly six months ago he noticed a little hard lump on the right breast. It has been an open sore nearly six months. It is exceedingly hard, feeling like a piece of wood let into the skin. The edge is very well marked, and there is a certain amount of inflammatory redness about it, which I connect with the obviously septic nature of the ulcer. On examining the axilla I find there are one or two small hard glands. There is no nipple on the affected side. You will notice also that this ulcer is not raised very much: there is no prominent fungating mass. On his right leg, just below the knee, he has other scars, and I think the question has therefore been raised whether the condition on the chest is tertiary syphilis. But I think there can be no doubt that this is a malignant growth, and the question is whether it is epithelioma of the nipple or scirrhus of the breast. I think it is scirrhus and not

Digitized by Google

epithelioma, chiefly on account of the absence of any prominence of the growth, and partly also owing to the fact that scirrhus of the mammary gland is much more common than epithelioma of the nipple. The latter would probably be in the form of a prominent button-like growth. It therefore seems clear to me that it is a scirrhus of the male breast. When I put the pectoralis major on the stretch I find the growth is absolutely fixed to the muscle. It does not move very freely even upon the ribs or intercostal muscles. I feel above the clavicle, and find nothing there. The other breast has nothing in it, and as far as I know there is no disease in any other part of the body. It is a very bad case. Carcinoma of the male breast is worse than that disease in the female breast. If you have two carcinomata of the same size, one in a man and the other in a woman, the one in the woman is far better suited for operation, because in her case you can cut away the growth much more freely. In this man not only is the skin extensively involved, but the growth is firmly attached to the muscle and to the fascia over the intercostal space. The man's general health does not appear to be affected, the glands in the axilla are very slightly affected, and, as far as I know, he has no other disease. I think removal ought to be performed, but I do not regard the prognosis as at all favourable.

Extensive Carcinoma of Bladder and Rectum.

The next patient is a man æt. 60, who complains of trouble with his water. He says it is two years since he was quite well. The first thing he noticed was that he could not pass his water freely, and he has passed blood in it. He comes with a discharging sinus near the umbilicus, which he has had for three weeks, and you at once become aware of a foul discharge from it. It is discharging brown fœtid material, but I have not quite made up my mind whether the discharge is fæcal, or urinous, or purulent simply. As my hour is almost at an end, I have not time to go into all the points of the case. Three weeks ago the swelling formed and then broke. I think it very likely that the sinus is connected with the intestine, or at any rate with the bladder, possibly with both. On examining the abdomen one feels at once a great hard round mass, dull on percussion, and of about the size and shape of a gravid uterus at six months. There is nothing else apparently wrong with the abdomen. I have examined him by the rectum, and I can feel a great mass between my finger in the rectum and my hand on the outside. It is very slightly movable. In the rectum, as high up as my finger will reach, there is a ragged ulcerated surface on the anterior wall, evidently in connection with this place.

I have not time now to say much more about this case, but my conclusion is that the tumour is a large mass of carcinoma, which originated presumably in the bladder, because the bladder was affected first, and there has been hæmaturia. I think it has gone through into the rectum; it has broken down and is now discharging the broken-down material. One can of course find out more about the case by examining the discharge—testing whether it is fæcal matter or urinous. Possibly the growth began in the rectum and then involved the bladder. It is more common for large growths like this to begin in the bladder than in the rectum. When they start in the rectum they usually produce more marked rectal symptoms. His rectal symptoms are comparatively slight, but the bladder symptoms were the earliest, and they have been the most severe. No treatment can be attempted here. The question may be raised whether it can be one of those retro-peritoneal tumours ulcerating into the bladder and the rectum. But I do not think it is one of them.

Epithelioma of Soft Palate and Tongue.

This man, who is forty-eight years of age, came to me at the Royal Free Hospital a few days since saying that three or four months ago he noticed something wrong with his mouth, and had some little trouble in swallowing. He did not seek advice for it until last Monday. He is a healthy-looking man, not having anything obviously the matter with him. But on close inspection he is seen to have some fulness behind the right angle of the jaw. On looking into his mouth there is at first nothing obvious to be seen. He puts his tongue out to the right; in other words, there is something on that side which prevents him from protruding the tongue properly. On depressing the tongue I can see just the top of an ulcer, which presents almost the same characters as that ulcer which we saw on the breast, and it also has almost the same feel. It is an epithelioma of the

Digitized by Google

soft palate and base of the tongue, but it is very far back. Here is a mass near the angle of the jaw which is evidently an aggregation of epitheliomatous glands. He is quite well in other respects. [This extensive epitheliomatous mass was completely removed a few days later by Krönlein's operation with, so far, a most satisfactory result.]

Epithelioma of Soft Palate, Tonsil, and Tongue after Operation.

This man, æt. 62, illustrates almost exactly the same disease, and almost to the same extent, as the last one, and this patient came to the Royal Free Hospital six weeks before the last one. If I had shown him to you seven weeks ago you would have seen a case almost precisely similar to the previous case, except that the glands were not so much involved. This growth involved the tonsil, and extended from the middle of the soft palate to the tongue; it was about as unfavourable an epithelioma as you could imagine. The two favourable points were that the glands were but little affected and the man's general health was good. I did a large operation (Krönlein's operation) for the removal of it. You see how far I split the cheek back to get a good view of the pharynx, and then I removed practically the whole of the soft palate and all the tissues involved. He did not lose more than three or four ounces of blood altogether. I did not touch the gland at that sitting, but left it for a subsequent operation. You will find a hard movable gland here, which will be removed on Friday next.

I am sorry that I have had to hurry somewhat over the last cases, but we have to-day had an unusually large number.

Analogy between Yellow Fever and Snake-bite.—The resemblance between yellow fever and the symptoms that follow the bite of a venomous serpent, suggests that the toxins may be identical. Especially remarkable is the degeneration of the liver-cells, much more rapid after a snake-bite than in yellow fever in which the toxins have first to be elaborated, while in the snake-bite they are introduced already manufactured.—*Revista Medica de Bogota, Colombia*, No. 251, N. Osorio, *Journ. A.M.A.*

A PATHOLOGICAL DEMONSTRATION.

At the Hospital for Sick Children, Great Ormond Street, June 6th, 1901.

By J. H. THURSFIELD, M.D.

LADIES AND GENTLEMEN,—The pathological material of a hospital like this naturally varies considerably, including both ordinary and rare specimens, and it is difficult to know whether to devote one's attention before a select audience to subjects which are rare or to those which are common. I think I have rather more of the rarities than of the common pathological specimens here.

I first want to direct your attention to this case of malformation of the œsophagus. It is an example of the only malformation to which the œsophagus is subject. This is the stomach of a child with a gastrostomy wound in it. The œsophagus, starting at the back of the pharynx in the ordinary way, ends in a blind sac about an inch below the larynx; then it will be seen that the lower portion of the œsophagus passes directly upwards from the stomach and enters the trachea just above its bifurcation, so that the œsophagus is in two portions. The clinical history of this child is interesting in this connection. It was a baby of four days old who had never succeeded in swallowing any food, and a soft catheter did not pass down the œsophagus. Therefore the surgeon who was called in to see him did a gastrostomy, and the child was fed for nearly a week through the gastrostomy wound. We did not diagnose the condition, because after the gastrostomy had been performed, and the child was fed with milk through the gastrostomy wound, he actually vomited by the mouth some of the milk which was put into the stomach, and the presumption is that that milk actually came from the stomach through the trachea into the back of the mouth.

This malformation is a developmental error, of which you will find a long account in the 'Pathological Transactions,' vol. xli, by Mr. Shattock, of St. Thomas's. The œsophagus and trachea are developed from the mesenteron. The posterior wall of the mesenteron becomes kinked, so that instead of going in a straight line there is a sharp bend inwards. As development goes on there comes to be at this point a communication between

Digitized by Google

the œsophagus and the trachea, and a complete interruption of the œsophagus,—the upper portion ending as a blind dilated sac, at the point where the lower pierces the wall of the trachea. I show you also a specimen of the same condition, which was mounted for the museum some time ago, while there are several specimens in the College of Surgeons Museum.

We now pass to another rarity. The other day we had a child sent down from the whooping-cough ward to the post-mortem room who had tubercle. Three days before his death he developed subcutaneous emphysema, which began in the neck and then became general. That may have accounted for the fact that a serious item in his pathological condition had been overlooked, namely, suppurative peritonitis due to perforation of the anterior wall of the stomach. You can see the perforation at the middle of the anterior wall, just below the lesser curvature. That is the only perforating gastric ulcer in a child of under ten years that I have heard of. Ulcer of the stomach is an extremely rare condition in children, but one does find it not very rarely in tubercular children. But a gastric ulcer of this appearance is almost certainly not tubercular—it is too clean cut and the edges are not sufficiently undermined; while there are no tubercles to be seen in the peritoneal coat of the stomach. We were unable to discover the origin of the emphysema in the chest, and it has been suggested that it was the perforating gastric ulcer, and that the effusion proceeded up the mediastinum, and became subcutaneous in the neck. But that explanation does not commend itself to me.

Next I would like to show you an instance of a rheumatic pericarditis. Here is a pericardium turned inside out. It is the heart and pericardium of a child nine years of age, who had had an attack of rheumatism previously, and came in with rheumatism and pericarditis, and died about a fortnight afterwards. You probably know that rheumatic pericarditis is much more common in children under twelve years of age than it is in adults; indeed, a considerable number of post mortems which take place here are associated with pericarditis. It is rare to find a heart from a rheumatic child, or one who has died of chorea, in which there is no pericarditis. But yet there is practically never pericardial effusion. Pericardial effusion is treated of in the text-books of medicine as if it were not an uncommon accompaniment of rheumatic fever, or, at any rate, of rheumatic heart disease; but I believe that the evidence upon which these statements are based is almost entirely clinical and not pathological. I have gone through the whole of the records of the post mortems here for the last twenty years, representing some seventy cases of rheumatic pericarditis, and, with only one exception, I have been unable to find any record of a large pericardial effusion. That one exception occurred twenty years ago, in a boy of eleven years of age, who had a large amount—about ten ounces—of serous fluid in his pericardial cavity. In the other remaining cases the greatest amount of fluid was under three ounces. In the present case there was in a pocket at the back of the heart, lying behind all the great vessels, about two ounces of rather blood-stained serum. So one must realise that to operate, either by paracentesis or by ordinary incision upon a case of rheumatic pericarditis, although on clinical grounds it may seem somewhat tempting, is unwise. You will almost certainly come down, as we should have done in this case, upon a pericardium united to the heart-wall by firm adhesions which, if they had been separated, would have bled freely. In fact, they did bleed post mortem, and there was evidence in the specimen of bleeding into these adhesions during life. We should have made the boy very much worse without getting anywhere near the fluid which was hidden at the back of the heart.

To compare with that I will show you another similar heart from a rather older child, a girl, with a pericardium so adherent that you can hardly separate the pericardium from the heart by means of a probe. That also is a case in which some time ago a discussion took place as to whether an incision should be made. You can see it would have been quite impossible to have known at the operation when one got into the pericardial sac at all. I might mention that Dr. Poynton, who has been working at this subject, has succeeded in isolating an organism by which he has been able to produce a very good imitation of rheumatism in rabbits, got some of his material from the first heart I passed round. He has isolated a diplococcus in, I think, seventeen cases, and has produced by it endocarditis, arthritis, and even rheumatic nodules in rabbits.

Digitized by Google

I now show you two hearts which illustrate a rare condition, namely, tubercle of the heart-wall. You can see in this specimen the tubercle grows at the anterior aspect of the heart. There is a large whitish-yellow nodule in the wall of the septum ventriculorum, and on turning to the inner wall of the ventricles you will see a large caseous mass attached by a pedicle which communicates with the nodule in the wall. The case is clinically interesting, because this caseous nodule was softening and discharging caseous material into the circulation. The child's history was that he was in his usual state up to twenty minutes before midnight; but at about that time he gave a loud cry. The nurse went to him, and then went out of the ward to get him something to drink, and when she came back he was dead. I suspected, although at the post mortem we could not prove it, that some of this caseous material entered one or other arteries of the brain, and produced death by embolism.

Tubercle of the heart-wall is a very uncommon form of tuberculosis. It usually occurs as miliary tubercle of the visceral pericardium, particularly round the origins of the great vessels. In the course of a year one generally sees two or three cases of that condition. But you have to look closely and examine carefully so as not to miss it. The other variety is, as in the present instance, a caseous nodule in the wall of the heart. That is a much rarer phenomenon in tubercle, but still it occurs sufficiently for some thirty or forty cases to have been reported. A paper was published by an American physician about two years ago, with an elaborate survey of the symptoms associated with tubercle of the heart-wall. But on the whole I think he failed to make out any case for supposing that these caseous masses in the walls of the heart had any relation to clinical symptoms. One of the chief points to be relied upon was an irregularity of the pulse. But in the last stages of tubercle it is not infrequent to get irregularity of the pulse. I do not think the matter is worthy of much attention.

With regard to tubercle of the valves I believe it does occur, but we have no specimen in the museum here, and I have never seen any specimen in which I could certainly say there is tubercle of the valves. But I see no reason why it should not occur. In the second specimen I show to-day the tubercle almost certainly began in the endocardium, and not in the muscle of the heart; and there is no particular reason why, if it begins in the endocardium over the muscle, it should not begin in the endocardium over the valve.

The next thing I want to show you is this specimen, from a post mortem to-day, of what is perhaps the most common form of tuberculosis in children —that is to say, a general caseous tuberculosis. It is a chronic disease, which may last three or four months, or even six months or longer, and post mortem it is always associated with an enormous distribution of caseous tubercle. You can see that the spleen is one mass of large caseous foci, both in the substance and in the capsule. In the lungs there is precisely the same condition in all the lobes, and also, what is more unusual, a patch of caseous pneumonia, which has some resemblance to an infarct in the upper lobe of the right lung. An additional reason for showing this specimen is that it shows very well the ring of enlarged cervical and tracheal glands which give rise to the cough which simulates whooping-cough. This child had a paroxysmal cough; he was not actually heard to whoop, but whooping-cough was suspected in connection with his case. This form of tubercle is associated with an irregular and often a hectic temperature. The hectic temperature, I believe, depends chiefly upon the degree of softening and infection by organisms other than the tubercle bacillus.

Another form of tubercle which in the ordinary text-books is not sufficiently distinguished is acute miliary tuberculosis. That is a condition absolutely distinct from acute general tuberculosis, or general diffuse tuberculosis. Acute miliary tuberculosis is an acute specific infectious disorder, which has perfectly recognisable clinical features and post-mortem appearances. Of course it is produced by the same organism, but it is practically incurable; one never sees these cases recover, and it is rapidly fatal, the cases not, as a rule, living beyond a month or five weeks. Here are some temperature charts which show how these cases progress. Here is the chart of a child who came in with nothing the matter with him except a little fever and some bronchitis. After he had been in ten days his temperature rose from subnormal to 104·6°, and from that moment his temperature never became regular again. That is the

Digitized by Google

kind of course which an acute miliary tuberculosis always pursues. Here is another. A child with tubercular hip goes wrong suddenly one day, his temperature rises from normal to 104·6, and from that moment till death, four weeks later, it never becomes regular again. Those charts show what I mean when I say it is an acute specific infectious disorder. You might perfectly well take such a temperature chart as indicating the onset of scarlet fever. The onset of acute miliary tuberculosis sets in with severe headache, sudden rise of temperature, severe constitutional disturbance, sometimes with sickness, sometimes with shivering fits. The great difficulty is that when you find these symptoms you find no other definite indications, and you hesitate for several days as to whether you are to consider it as a case of miliary tubercle or of some other infectious disorder. For instance, in the first case I spoke of, the moment the boy's temperature went up he was isolated for measles, and was regarded as a case of that disease for the first four or five days. He had had coryza, but he never developed a rash. The diagnosis of acute miliary tuberculosis was at last confirmed by the appearance of miliary tubercles in the choroid. The surgical case also was isolated, and she was supposed to be going in for scarlet fever. Here is the lung of the boy to whom the first temperature chart belongs. It is not a typical example of miliary tuberculosis, because there are some caseous patches in the lung; but the probability is that those caseous patches were the actual source for the dissemination of the tubercle throughout the body. But if you ignore those patches and look at the rest of the lung you will see it is a very fair example of the post-mortem appearances of acute miliary tuberculosis. Alongside of it I will put this case of acute caseous tuberculosis, running quite a different course, and having a different post-mortem appearance from the other one. A large proportion of the children who have acute miliary tuberculosis develop choroidal tubercle. This eye shows the tubercle of the choroid from the first of those two cases. Of twenty-five cases of tubercle of the choroid, twenty were found to be from subjects of acute miliary tuberculosis.

Apart from those forms I have shown you there are the chronic forms of tubercle. Here are the lungs from a case of very chronic tubercle, whose clinical history was rather interesting. If you separate the lungs you will find cavities in many places. The boy was one year and seven months old. There is one cavity between the lower lobe of the lung and the diaphragm, and another cavity opening into the pleura, with a smaller cavity at the apex of the lobe. There is a coarse tubercle filling up the whole of the rest of the lungs. The history is that the child was in the ward for many weeks slowly wasting, and that he was thought to have an empyema. He was punctured once, but nothing was found. A week later he was punctured again, and a syringe full of pus was withdrawn from the axilla. A rib was resected and the pus evacuated, but the boy died three days later. Post mortem, it was found that the cavity on the lateral aspect of the right lung communicated with the pleural cavity. That leads me to say something about tubercular empyema. There seems to be a very general impression abroad that a large proportion of empyemata in children, or in fact in adults, are due directly to tubercular infection. I am certain that the exact opposite is the case, namely, that a very small number of empyemata have anything to do with tubercle. The grounds for that belief are, in the first place, that the vast majority of children with empyemata here have their empyemata following directly upon an attack of broncho-pneumonia, and the vast majority of empyemata grow pure cultures of the pneumococcus; some four to six per cent. grow the streptococcus; and one or two per cent. grow staphylococcus or mixed growths. I have tried many times to find the tubercle bacillus in likely cases, but I have never yet found it in the pus of an empyema. The only way in which one could exclude the tubercle bacillus with any certainty would be to inoculate guinea-pigs with the pus. But even then the pyogenic organisms would probably vitiate the result by destroying the animal before the development of tuberculosis. Going to post-mortem records, it is obvious that if an empyema is tubercular there are considerably greater chances that the child will die than that it will recover, and yet out of the fatal empyemata here in this hospital only 16 per cent. are associated with tubercle, that is to say, in the post-mortem records here for twenty years. That is, 84 per cent. of the empyemata dying in this hospital are found to have no tubercle whatever in their

Digitized by Google

bodies. On the other hand, of the cases of children who die with thoracic tubercle in their body at the time of the post mortem, some 1400 cases in twenty-five years, the percentage of empyemata is rather over two; so that the association of empyema with tuberculosis of the lungs is very slight indeed. You must recollect in relation to these figures, first, that the tubercular empyemata are more likely to prove fatal than non-tubercular, and therefore probably we get a larger percentage of empyemata dying which are associated with tubercle than of others. If one could ascertain equally well in the cases that recover the condition of the thoracic organs, that 16 per cent. would, I think, come down to nearly 2 per cent. or 3 per cent. Secondly, you must recollect that in these figures I have taken no account of cases which, like this, were practically moribund. It is quite obvious that if you get a tubercular cavity which is open to the outer air bursting into the pleural cavity you will get pyogenic organisms from the outer air or the pneumococcus from the lung invading the pleural cavity, and setting up suppuration. That, to my mind, is the only way in which a tuberculosis of the lung ever leads to empyemata. In all the cases which I have had anything to do with personally, in which tubercular disease has been found associated with empyema, I have been able to cultivate from the pus of the empyema the pneumococcus and other pyogenic organisms. This latest case grew pneumococcus, staphylococcus, and streptococcus, but there were no tubercle bacilli present in the cultures at all, nor in the films.

There is another form of tuberculosis which is very well illustrated by this specimen. That is, the very chronic glandular tubercle in which the glands at the root of the lung invade the lung and set up a local tuberculosis. In this specimen the tubercle is principally confined to the upper lobe, which has been invaded by one of the caseous glands at the root of the lung. That is a form of tubercle which is usually extraordinarily chronic, and which I believe accounts for a certain number of the cases of consolidation at one or other apex which, after lasting for many weeks, recovers. That means that the tubercle is encapsuled and becomes shut off from the surrounding tissues, and you get no more bother with it until something else lights it up again.

I show you also miliary tubercle of the spleen, which is quite distinct from the tubercle in the specimens I have already passed round. In this you can scarcely see the lesions of tubercle which are so finely dotted about.

Here is a specimen of tuberculosis of one of the long bones, resembling a tubercular dactylitis, tuberculosis of the left tibia, for which the child was brought to the hospital. It is a very unusual condition in any of the long bones. Of course in the phalanges of the hands it is not at all an unusual condition in children, particularly in children of under four years of age. This child was ten years of age, and the tuberculosis was apparently quite stationary. He died of acute tubercular peritonitis.

Next I show you a tubercular spleen, and in it a tubercular infarct. In addition to the other small caseous patches in the spleen there is this big caseous patch at the upper end, which is a very unusual condition. I have not seen anything like it previously, and various other pathologists to whom I have shown it are equally unacquainted with the condition. I think there can be no doubt that it is a very recent tubercular infarct of the spleen.

It is extraordinarily difficult to get a good specimen of tubercular meningitis for museum purposes, and now this specimen is put up I am not altogether satisfied with it. But on the whole I think it would be difficult to replace it with a specimen which shows the tubercles better, because of the hæmorrhage which has taken place. A typical tubercular meningitis is principally a basic meningitis, whereas this specimen shows an immense number of small tubercles scattered all over the hemisphere, particularly the hemisphere where the hæmorrhage has taken place. Even in a fresh specimen it is very hard to find typical tubercles at the base of the brain, and one often judges of their presence by associated conditions. I think tuberculous disease of the convexity much more common in tubercular meningitis than the books teach us.

I also show you a specimen of sarcoma of the lung in a child, the original disease being sarcoma of the kidney. A happy feature about this malignant disease in children is that the patients do not suffer; they get fat and healthy and look comfortable. This particular child was found, on the post-mortem table, after six months' residence in hospital, to be fat and well-conditioned, and the only trouble she really had during her life was from the attacks of bronchitis, presumably caused by these glands and masses of malignant growth within the thorax.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 461. WEDNESDAY, AUGUST 28, 1901. Vol. XVIII. No. 19.

CONTENTS.

PAGE

*A Clinical Lecture on South African Dysentery. By J. W. WASHBOURN, M.D., F.R.C.P. 289

*A Surgical Consultation at the Medical Graduates' College and Polyclinic. By E. W. ROUGHTON, B.S., F.R.C.S. 294

*The Practice of Blood-letting. By J. F. BRISCOE, M.R.C.S. (*concluded*) ... 299

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

SOUTH AFRICAN DYSENTERY.

Delivered at Guy's Hospital, July 17th, 1901,

By J. W. WASHBOURN, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—I have chosen dysentery as the subject for my lecture to-day for several reasons. I have seen a large number of cases during the last eighteen months in South Africa; the disease occurs to a not inconsiderable extent in England at the present time; and you will probably meet with a number of cases among soldiers and others returning from South Africa.

We have at the present time two cases of dysentery in our Clinical Ward. I will read you brief notes of these cases:

CASE 1.—S. J. O—, late Corporal in the Imperial Yeomanry, admitted May 29th. He went out to South Africa in March, 1900, and almost as soon as he arrived in Cape Town the whole of his Company, himself included, went down with "dysentery." The patient got free of this in about three weeks. When in Pretoria, in June, 1900, he had an attack of enteritis which lasted three weeks, and which he attributed to eating bad oysters. In September he again got dysentery, which lasted about a month, but did not prevent him getting about. He was then invalided home, and on the voyage soon got quite well, the dysentery completely disappearing. He arrived home in December, and kept well until the beginning of April of this year, when he commenced to suffer from alternate attacks of diarrhœa and constipation. On May 25th he was seized with a sudden attack of diarrhœa with pain in the hypogastrium. On the following day the bowels were open ten times, and this continued until admission into the hospital on May 29th. His motions have been liquid, sometimes streaked with blood, and at times quite large quantities of blood have been passed. Since admission he has been carefully treated by dieting

Digitized by Google

and drugs, and has gradually improved, and is now almost completely well.

This is a typical example of a patient who has suffered from acute dysentery, which has become chronic. In considering the case you will notice that the patient states that the whole of his Company suffered with dysentery immediately after arrival, and that he had several attacks subsequently. The probability is that the first attack was one of gastro-enteritis, from which almost everybody suffers after landing in South Africa. True dysentery rarely attacks such a large number of people at the same time.

This gastro-enteritis is of some interest. Almost everyone is attacked. When the Imperial Yeomanry Hospital reached Deelfontein, the medical officers, sisters, and orderlies, were, with hardly an exception, attacked with this complaint. The symptoms are an acute diarrhœa, usually painless, but sometimes attended by colic; there may be vomiting, but this is not common. The temperature may reach 103° or 104°, but rarely continues raised for more than twenty-four hours. As a rule the patient recovers in a day or two, but the diarrhœa may persist for a week or longer. I have no doubt but that this disease is quite distinct from dysentery. The stools consist of liquid fæces, and only occasionally contain a small amount of blood. The disease may be prevalent at a time when no true dysentery occurs. It is seen especially in the hot weather, and appears to be analogous to the summer diarrhœa noticed in England. Patients become ultimately acclimatised, and after one or two attacks are no longer affected. The treatment consists in the administration of a dose of castor oil with a little tincture of opium and absolute starvation for twenty-four hours.

Case 2.—J. T. R— was admitted on June 10th, 1901. About two and a half years ago he fell forty-five feet and struck his right side, and was laid up for several weeks. Two years ago he suffered from dysentery in Alexandria, which lasted about three weeks. On April 28th of this year he slipped off a plank and fell on to the deck a short distance, jarring his back and loins, but the pain passed off in a few minutes. The following night he felt a sudden sharp pain in the right side and shoulder. He was sent into a hospital, where he remained till May 31st, when he came to London and was admitted to this hospital on June 10th. The pain has been continuous ever since its commencement. On admission the patient was found to be a healthy-looking man with a temperature of 101°, pulse 116, and respirations 28, complaining of great pain in the right side and right arm. On examination there was slight bulging near the costal margin in the right anterior axillary line. There was dulness at the base of the right chest. The edge of the liver could be felt below the costal margin, and the abdominal muscles in that region were rigid. There was considerable tenderness over the liver. On June 13th a needle was put into the chest over the dull area, but nothing was withdrawn. The patient gradually got worse and on the 18th was delirious, the temperature remaining high. On June 20th the patient was put under an anæsthetic, and an abscess was discovered in the liver, which was opened and a drainage-tube inserted. The pus which was removed was of a chocolate colour, and on examination was found to be sterile; no amœbæ were found. The patient is gradually improving, but the abscess is still discharging the same chocolate-coloured pus.

This case is an example of a liver abscess following upon dysentery. I will now give you a description of dysentery as it occurs in South Africa, and I will describe it under several types.

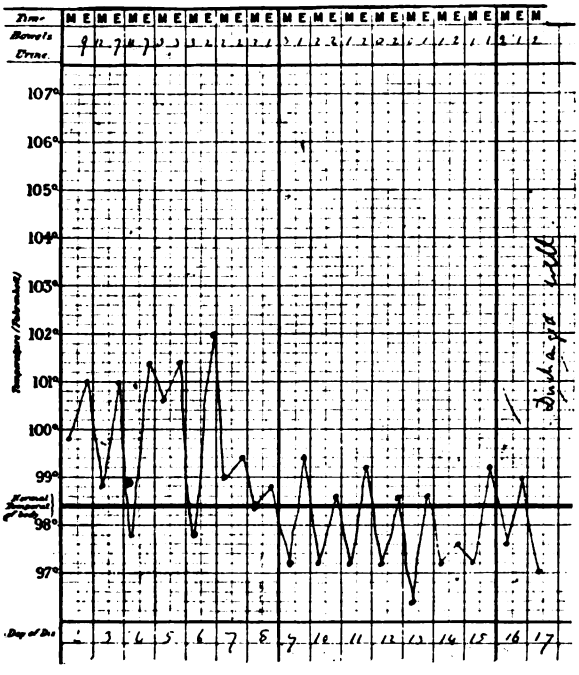
Type I.—This is the most common type. It frequently begins with an acute attack of ordinary diarrhœa, which, however, is quickly followed by the passage of blood and slime. Sometimes the passage of blood and slime is the first symptom of the disease. The bowels are very frequently opened, and there is marked tenesmus. The patients are sometimes hardly ever off the bed-pan, constantly feeling the desire to go to stool, but on each occasion only passing a small quantity of blood and slime. There is abdominal pain, and tenderness along the colon. The characteristic feature of this type is pyrexia, which lasts about six days (*vide* Chart 1). The pyrexia may last a shorter or a longer time than this, but the usual duration is six days. This pyrexia is so characteristic as to suggest that dysentery is a specific disease, modified by the affection of the intestine. As the fever subsides the motions become less frequent, and the fæcal matter begins to appear. In three or four days the fæces again become normal. The whole disease lasts about nine days.

Digitized by Google

The tongue is somewhat characteristic; it is almost always covered with a white, moist fur, looking as if it had been covered with a thick layer of paint.

This is the usual course of an attack, but in some cases the disease ends fatally. Death may occur during the pyrexial stage, the patient becoming collapsed, and the pulse failing. Death more frequently occurs at a later period, some nine or ten days, or longer, after the fever has subsided. The tongue becomes red and glazed, the patient becomes very wasted and collapsed, with feeble pulse and blue extremities. A marked feature frequently is retraction of the abdomen. The mental condition, as a rule, remains good to the

CHART 1.



end, and the patient frequently does not recognise the gravity of his condition. The stools may consist of blood and slime to the last, or they may contain liquid fæcal matter.

Instead of recovery or death the disease may become chronic, and then the patient suffers from diarrhœa, with, at times, passage of blood and slime, this condition frequently alternating with constipation. The motions contain hard lumps of fæces, often covered with blood and slime. This is the condition of the patient in Case 1, whose report I have already read to you.

The course of a chronic case is not infrequently interrupted by relapses, with symptoms similar to the primary attack, viz. pyrexia and the passage of blood and slime.

TYPE II.—*The typhoid type.*—In this type the disease begins in the ordinary way, with the passage of blood and slime The pyrexia, however, does not subside at the end of the first week, but continues for two or three weeks longer, as you will see from Chart 2, and the patient falls into a condition very similar to that met with in typhoid fever. The spleen is sometimes enlarged, and the blood and slime often disappear from the stools, and are replaced by liquid fæcal matter. The motions are brown, liquid, and sometimes contain small sloughs, and are very offensive. There is sometimes constipation lasting for several days. The abdomen is often retracted, which is an important sign in distinguishing the condition from typhoid. Sometimes neither blood nor slime are passed during the whole of the illness, and the intestinal symptoms are quite in the background.

These cases are, I believe, due to a secondary septic invasion of the tissues, and I believe them to be analogous to the typhoid form of scarlet fever.

TYPE III.—*Mild cases.*—These are cases in which the attack lasts for a few days. There is the usual passage of blood and slime, but no pyrexia, and recovery rapidly ensues.

COMPLICATIONS.—Perforation frequently occurs in the fatal cases; I have known as many as six perforations to occur in one case. On account of the previous matting together of the intestine, the ensuing peritonitis is more or less localised, and the symptoms are consequently not so characteristic as they otherwise would be.

Hepatitis, with enlargement and tenderness of the liver and slight pyrexia, now and then occurs. This may result in abscess, but is decidedly rare as compared with some forms of tropical dysentery. At Deelfontein in 250 cases abscess occurred only once.

I have seen purpura now and then associated with dysentery, but I do not think there was any causal relationship between the two.

I have seen cases of enteric and dysentery existing at the same time, and in fatal cases the two lesions have been found side by side. In other cases patients recovering from dysentery have con-

Digitized by Google

tracted enteric, or patients suffering from enteric have contracted dysentery during convalescence.

MORTALITY.—The mortality is not high. Of 379 cases admitted into the Yeomanry Hospital at Pretoria there were seven deaths, a mortality of 1·86 per cent.

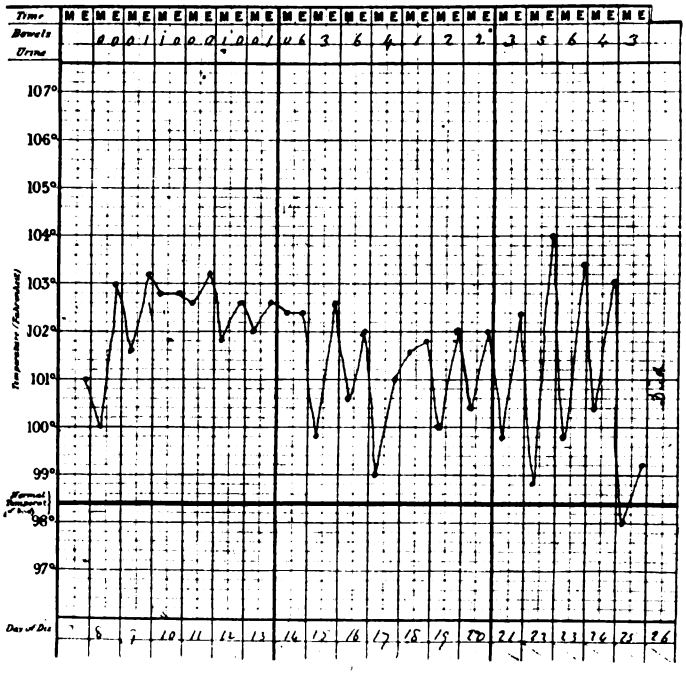
POST-MORTEM APPEARANCES.—The lesions are chiefly confined to the large intestine, but the last two or three feet of the small intestine are sometimes involved. The wall of the intestine is thick and infiltrated, and is often studded with petechiæ. In all the cases I have seen ulceration has also occurred. The ulcers may be small and punched out with clean-cut edges, or the edges may be ragged and the ulcers larger, with the floor consisting of a yellow slough. In some cases almost the whole of the mucous membrane is destroyed, and the intestine has a honeycombed appearance, with only small islets and cords of mucous membrane remaining.

In all cases the rectum and sigmoid are the most severely affected, and next in severity comes the cæcum. Sometimes they are the only parts affected, but not infrequently the whole of the large intestine is involved.

In the small intestine I have only observed thickening and infiltration, with sometimes a few small ulcers.

PATHOLOGY.—At present we know very little about the pathology of this disease. I have frequently examined the evacuations, but have never found amœbæ. The slime on microscopical examination is found to consist of a very large number of leucocytes, a few large cells which are probably macrophages, and sometimes a considerable amount of intestinal epithelium. The large cells may be mistaken for amœbæ, but they can be distinguished by the fact that they possess no active amœboid movements. In the one case of abscess of the liver which I carefully examined, the pus immediately after removal contained a large number of bacteria consisting of streptococci and a bacillus belonging to the coli group. It, however, contained no amœbæ.

CHART 2.



DIAGNOSIS.—Dysentery as a rule is quite easy to diagnose. One must remember, however, that here are other conditions which may give rise to the passage of blood and slime—for example, cancer of the rectum, intussusception, or a polypus. We must also remember that the blood may come from hæmorrhoids. These conditions, if remembered, can be readily distinguished. I have

Digitized by Google

known chronic constipation with fæcal accumulation mistaken for dysentery. Patients state that they are passing blood and slime, and on examination this is found to be due to scybala either in the rectum or in the descending colon. As scybala are not infrequently passed in chronic dysentery a mistake may be easily made. Careful inquiry into the history of these cases should, however, clear up the diagnosis.

The typhoid type of dysentery is often difficult to distinguish from typhoid fever. In one case where neither slime nor blood was passed, and the patient died at the end of three weeks from perforation, the diagnosis was only cleared up at the post-mortem examination. The enlargement of the spleen which sometimes occurs renders the diagnosis more difficult. However, in most cases there is something by means of which the diagnosis can be correctly arrived at. There is usually at the commencement blood or slime in the motions, the abdomen is not infrequently markedly retracted, and the motions may be exceedingly foul from a gangrenous process in the intestine. An examination of the blood for the agglutination test is often of great value. The most difficult cases are those in which enteric and dysentery are present at the same time.

Treatment—In the acute stage absolute rest in bed is essential. The patient should not be allowed to get up even to use the commode. The diet should consist of milk, unless there is any evidence that it is not being digested or that it disagrees. In such cases meat juice, whey, etc., should be substituted for the milk.

There are two specific modes of treatment. One is magnesium or sodium sulphate, and the other is ipecacuanha. In many cases the results of either form of treatment is most striking, and sometimes when one form of treatment has failed the other has succeeded. Magnesium sulphate is administered in drachm doses every hour until the motions assume a fæcal character. If after twenty-four hours no effect is produced, the treatment had better be left off. Ipecacuanha is administered in the following way: After two or three hours abstention from food, a small dose of tincture of opium or chlorodyne is given, and half an hour later, thirty grains of powdered ipecacuanha is administered. In three or four hours the dose is repeated. The object of this mode of administration is to prevent the patient being sick. I have frequently made observations upon the value of deemetinised ipecacuanha, and I feel sure that it is not so good as the crude powder. Should the diarrhœa continue after the admistration of the magnesium sulphate or ipecacuanha, a mixture containing bismuth and morphia, or some other astringent, should be given; but care must be taken not to administer these drugs too freely, otherwise constipation may ensue. Where there is marked tenesmus, morphia suppositories have often a wonderful effect. In some cases where other treatment has failed, the subcutaneous injection of morphia is remarkably efficacious.

Chronic cases are treated with opium and astringents in the same way as cases of chronic diarrhœa. Large rectal injections containing several pints are sometimes of value; the injection may consist simply of warm water, but it is usually advisable to add some astringent, such for instance as nitrate of silver, half a grain to the ounce. Careful dieting is of great importance, and meat should be avoided for a long time. As regards prophylaxis, chill should be guarded against. I have no doubt that a chill is an important predisposing cause, and that the wearing of a cholera belt is a safeguard against dysentery.

Relationship of South African Dysentery to Other Forms of Dysentery.

I have already mentioned that dysentery occurs to a not inconsiderable extent in England. I was referring to the dysentery which occurs in almost every asylum. There is a careful description of this form of dysentery in a report by Dr. Mott and Dr. Durham to the London County Council. From their description, I have no doubt that the disease is identical with the one which I have already described to you. There is the same fever and other symptoms. The post-mortem appearances appear to be identical, with the exception that acute cases sometimes die before ulceration has occurred—a condition which I have not observed in South Africa.

I have no doubt that the prevalence of dysentery in asylums is due to the same causes as its prevalence among troops on active service, viz. insanitary conditions. I believe that South African dysentery is infective, and this has been shown by

Digitized by Google

Dr. Mott and Dr. Durham to be the case with asylum dysentery.

In addition to these cases of asylum dysentery, sporadic cases have from time to time been described in England by Dr. Hale White and others, under the heading of simple colitis and ulcerative colitis. I am inclined to concur with Drs. Mott and Durham in their view that these cases are simply sporadic cases of the same disease as is met with in asylums. The cases which I myself have seen have closely resembled the typhoid form of dysentery which I have described.

Another form of dysentery is described under the heading of tropical or amœbic dysentery, and this form appears to be distinct from the South African type. The course of the disease is more prolonged, and liver abscess is a common result. In the stools and in the pus from the abscess amœbæ are found. Certainly in the South African dysentery amœbæ are not present and liver abscess is decidedly rare.

The Action of Electric Current of High Tension on the Human Body.—Brustein, after reviewing the literature on injuries caused by the electric current of high tension, reports the case of a man, thirty-nine years old, who, while working on an electric transformator standing on a perfectly dry floor, received a shock of 3000 volts., with 100 interruptions per second. He lost consciousness for twenty to thirty minutes, when he was able to get up and walk to the carriage. For about two months he was treated for the burns which he received, and recovered sufficiently to resume his work. Soon after he developed a neurosis associated with hysteria and neurasthenia. He presented a concentric narrowing of the field of vision, clonus of the patella, irregularity of the pulse, increased reflex action and muscular irritability, vasomotor disturbances and weakness of the extremities; in other words, this was a typical case of traumatic neurosis developing several months after the injury. The patient finally recovered under bromides, massage, and electricity.—*Vratch*, April 21st, 1901, vol. xxii, No. 16; *The Philadelphia Med. Journ.*, July 27th, 1901.

A SURGICAL CONSULTATION AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

June 5th, 1901.

By E. W. ROUGHTON, B.S.Lond., F.R.C.S.,
Surgeon to the Royal Free Hospital.

GENTLEMEN,—The Royal Free Hospital being situated close to the Midland, Great Northern, and Metropolitan railways, a comparatively large number of severe injuries are brought there, and many of these are of the lower extremity. Hardly a week passes by without the surgeon on duty being summoned to see a case of severe injury of the lower extremity, and very often it is found that amputation is necessary. On being called to such a case, the first question that presents itself to one's mind is whether an amputation will be necessary or not. In deciding that question one has to consider not only the local but also the general condition of the patient; such elements as age, general constitution, and habits, particularly with regard to alcohol, must all be weighed.

This patient is a good example of a very severe injury to the lower extremity, in which we were able to save the leg, although at first sight the limb appeared to be hopelessly mangled. The accident happened last July. He and some other men were engaged in taking a crane to pieces, when the chief parts came asunder prematurely, and the boiler fell across his legs, breaking both femora at about the middle, and at the same time crushing the lower part of his left leg to a very serious degree. The house surgeon, who was a man of very good judgment, examined the leg, and sent for me to perform amputation. Certainly when I first saw the limb I thought it would have to come off. But on more careful inspection I found that, although there was a comminuted fracture of the tibia and fibula three inches above the ankle-joint, and although the muscular bellies were protruding and much lacerated, and the skin extensively torn and stripped up, yet the main vessels supplying the foot seemed to be intact, and the circulation was going on briskly. As he appeared to be a perfectly healthy man, I decided to make an attempt to save the limb. On enlarging the wound to examine the nature of the fracture more minutely, I found that a piece of

Digitized by Google

the tibia about an inch and a half long had been knocked clean out of the bone, and embedded in the neighbouring muscles. As it was completely detached from all sources of blood supply, I thought it best to remove it. The fragment was not square, but longer on the outer aspect than on the inner; so when I had removed it I had two spiky ends of tibia to deal with; having sawn off the apex of each I managed to get in a couple of wires, and bring the two fragments into fair apposition. Having thus shortened the tibia, I had to take off a piece from each fragment of the fibula to make the two bones about the same length. During the operation I noticed that the anterior tibial artery was laid bare nearly the whole way to the ankle, and in this situation it appeared to be bleeding. But on closer examination I found that the blood was coming from a tiny branch which had been torn off close to the artery. I was very anxious not to tie the anterior tibial, as I could not be sure of the condition of the posterior tibial artery. Clearly if both these vessels had been obstructed, the vitality of the foot would have been so seriously interfered with that gangrene would have certainly ensued. I managed to get hold of this tiny vessel and tie it without interfering with the main vessel. The after-progress of the case was uneventful; it pursued an aseptic course, and there was no rise of temperature or suppuration; in the course of time it healed soundly, and is now a useful leg, although, perhaps, not a particularly elegant one. Luckily for him there has been more shortening of the right femur than of the left, so that he is not so lop-sided as he would otherwise have been. He tells me he gets on with his work all right, and finds no difficulty with regard to his legs. You see that he walks very well.

In many cases of this class a part of the limb has been taken off completely, or is so hopelessly smashed that there is no possibility of saving it. Here, again, there are questions of interest and importance to consider. It does not follow that because a limb has to be removed, the sooner it comes off the better. Such an idea is quite a mistake. I often see cases of severe injury in which the question of immediate amputation is not to be entertained at all. Sometimes a man is so terribly injured that the only thing to do is to leave him alone to die. I remember a case of severe compound comminuted fracture of both femora, in which the shock was so intense that the question of amputation, or, indeed, of any other treatment was not to be entertained, and we could only leave him to die. I remember another case of a man who came in with both feet torn off. He was in a very severe state of shock, and if an amputation had been done at once the result would, I feel sure, have been fatal. I am never in a hurry to amputate in these cases. I always prefer to wait an hour, or two hours, or even longer, until they come out of the primary shock; one can then do the amputation with success, whereas if it were done at once the additional shock of the operation would probably be fatal.

We have next to consider the position of the amputation, and the method of cutting the flaps, our object being to produce a stump which will carry an artificial limb to the greatest advantage. Our text-books of operative surgery describe many amputations which the artificial limb-maker regards as thoroughly bad. These are chiefly disarticulations and amputations within a few inches of a joint. As a general rule an amputation should not be done nearer the knee-joint than five inches, since an efficient joint mechanism in the artificial limb cannot be compressed into a smaller space. The "seat of election" should be just above the middle of the leg, instead of a hand's breadth below the knee, as usually given in text-books.

In fashioning the flaps we must adapt our method to the nature of the injury, and must be prepared to utilise any aspect of the limb where the tissues are uninjured. The garment must be cut according to the cloth. When there is plenty of material available I prefer to amputate by anterior and posterior flaps, and circular division of muscles. I operated by this method in the patient I now show, and I invite you to criticise the stump. You will observe that there is ample covering for the ends of the bones, the tissues over which are mobile and non-adherent. The scar is not terminal, but well towards the back, and out of the way of pressure. He is able to bear a certain amount of pressure on the end of the stump, but the bulk of the weight of the body is supported by the cup-shaped end of the artificial limb which receives the thigh. Notice how well the man walks; a casual observer would never guess that he had lost a limb.

Digitized by Google

The next case I show is one in which the tissues on the anterior aspect of the limb were too much damaged to allow one to cut an anterior flap. In this case I amputated by equal lateral flaps. The stump is a good one, but the scar is of necessity terminal.

I now show a man whose leg I amputated by Farabeuf's method, *i. e.* by a single large external flap containing all the structures right down to the interosseous membrane. This, as you see, makes a good stump, with the scar well to the inner side.

These two men have not yet got their artificial limbs, but I see no reason why they should not walk as well as the man who has just gone away.

The next patient is a man æt. 28, who had a perforated gastric ulcer. There was a history of dyspepsia for two years. About the 1st of January last the pain and the vomiting became worse, and he was laid up for a few days. During that time he noticed that the pain was less when he was lying on his back than in any other position—a point always suggestive of gastric ulcer on the anterior wall of the stomach. On the 8th February he had breakfast at four o'clock in the morning. He commenced his work at five, and at that time he was suffering from pain between the shoulders. At eight o'clock he vomited a pint of fluid, and the pain was so bad that he discontinued work for a short time. At 10.15, however, he was seized with a sudden severe pain in the epigastric region. I saw him at one o'clock in the afternoon. He was then complaining of much pain in the abdomen, but the expression on his face was not of that intensely anxious description which one so often sees in these cases. His pulse was 88 on admission, and an hour afterwards it was 116 per minute. The symptoms of collapse were only moderate. On examining the abdomen we found it intensely hard all over. Tenderness was present in every part, but there was no distension. The pain was localised, chiefly near the umbilicus. Liver dulness was present, and perfectly normal. I had no hesitation in diagnosing the case as one of perforated gastric ulcer, and I advised the patient to submit to operation immediately. The operation consisted in opening the abdomen by a vertical incision, the scar of which you see here. On opening the peritoneum it was noticed that no gas escaped. In perforation of a hollow viscus you often find free gas in the peritoneum, but in this case there was none. Presumably when the perforation occurred there was no escape of gas from the stomach. On opening the peritoneum I lifted up the liver, and on the anterior surface of the stomach I found some greyish-white fluid, and on sponging that away some more of the same sort was found to be welling up through a circular aperture one quarter of an inch in diameter. This aperture was surrounded by a thickened ring of tissue the size of half-a-crown on the anterior wall of the stomach, a third of the distance from the lesser to the greater curvature, and nearer the cardiac than the pyloric end. That is the position in which one usually finds perforating gastric ulcers in women. In men they are more often towards the pyloric end.

Next, I divided the left rectus transversely. That is a thing I always do without hesitation in an abdominal case in which I want more room for the manipulation. A branch of the superior epigastric artery required clipping. The area of operation having been surrounded with sterile gauze packing, four silk Lembert stitches were inserted close to the margin of the perforation, through the thickened tissues. Another tier of seven Lembert stitches were inserted through the surrounding healthy tissues, and by this means the perforation was effectually closed. Then the peritoneum was cleansed by sponging. This is an important point. Some authorities advise sponging, others recommend flushing. One cannot adopt the same practice in every case; the method employed must depend upon the extent to which the peritoneum has become soiled. If the peritoneum has been soiled locally as in this case, it is best to mop away the material with sponges, for by flushing one disseminates the septic material over the peritoneal cavity. But if, as in some cases, the septic material has already become generalised, sponging is a hopeless procedure, and one must flush. For that purpose it is desirable to use the most unirritating fluid.

In this case we anticipated that the result would be successful because the degree of collapse was slight, and the escape of the stomach contents had been strictly limited, so that there was only a local soiling of the peritoneum, not a general one. He made an uninterrupted recovery, with the exception of a stitch abscess.

Digitized by Google

By way of contrast to this simple, straightforward case, I might now briefly refer to some other cases in which the diagnosis was very difficult, and the treatment correspondingly unsatisfactory. The very next day a girl was brought to the hospital with symptoms of perforation, of only two hours' duration. I opened the abdomen, and rather to my dismay I found nothing except slight but generally diffused peritonitis. I examined both surfaces of the stomach, the vermiform appendix, the ovaries, and Fallopian tubes, with a negative result. She died the next day, and then we found—but not until we had removed the stomach—that there was a perforation on the posterior surface, near the lesser curvature.

About a week after that a deaf and dumb man was brought in, with a history of intestinal obstruction of three days' duration. The lower part of his abdomen was distended and tender, but in the region of the stomach there was no tenderness whatever. The liver dulness was normal. At the post mortem we found that he had a perforation on the anterior surface of his stomach, which nobody had diagnosed.

Another case was that of a girl who was brought in with typical symptoms of perforation of the stomach. She was much collapsed, and had a very small pulse of 130. I had no hesitation in diagnosing the case as one of perforating gastric ulcer, and I opened the abdomen. But I found nothing abnormal, and therefore closed it again. She made a good recovery, and it turned out afterwards that the symptoms were entirely due to eating pork sausages. She was seen by several other surgeons, and they agreed with me in the diagnosis of perforating gastric ulcer.

Here is a patient, a man æt. 47, who first found difficulty in swallowing solid food last November; the difficulty has steadily increased. For the last three months he has been unable to swallow any solid food. Since Christmas last he has lost two stone in weight. In a case like that one thinks of malignant disease of the œsophagus, and that is what I have no doubt he is suffering from. We passed a bougie, and it went down eleven inches from the teeth, but no further. As we could not get a tube into the stomach, the question of gastrostomy had to be considered. I strongly advised him to have this done before he got any worse. I think gastrostomy is not such a severe operation as the older text-books lead one to believe, provided it is done before the patient gets to a state of extreme weakness. In this case we did gastrostomy by Frank's method on April 30th, and during the time that the patient was being prepared for operation, and for a day or two afterwards, he was fed with nutrient enemata. The point about this method is that you make a valvular opening into the stomach. In the old-fashioned gastrostomy that most of you learnt about when students a large hole was made into the stomach, no attempt was made to establish anything like a valvular opening, and the contents of the stomach used to regurgitate almost as soon as they were put in, and the result was a large gaping wound, through which not only the stomach contents used to escape, but sometimes even folds of the stomach itself; the skin over the abdominal wall became excoriated and irritated, and the condition of the patient was not an enviable one. This operation is done by making a vertical incision through the outer third of the left rectus, in the position you see the scar. It is about one and a half to two inches long, going through the rectus muscle, separating the fibres rather than dividing them, until one comes to the posterior sheath of the rectus, which is divided and the peritoneum opened; the stomach is then sought for, seized with a pair of pressure forceps and drawn up into the wound; a stitch is put in through the abdominal wall and through the stomach at four points—above, below, and on each side. Then a second transverse incision is made, about one inch above the first incision, and the skin between the two undermined. A pair of pressure forceps is passed from the upper to the lower wound, and the apex of the cone of stomach is drawn upwards into the transverse wound, and fixed there by a couple of harelip pins. The lower wound is sewn completely up and covered with a collodion dressing. The stomach can be opened at once if necessary, but in this case it was not necessary to do so. I deferred opening it until May 2nd, the original operation having been on April 30th. I wish you to observe that he has a small opening through which the contents of the stomach do not regurgitate, and the skin around it is healthy. Since the operation he has not suffered from hunger, which was a very distressing symptom before. He is now beginning to gain in weight. Before the operation he weighed 8 st. 1 lb.; on

Digitized by Google

May 15th 8 st. 4 lbs.; on May 28th 8 st. 10 lbs.* The patient is provided with a silver cannula, which he can easily insert into the stomach; to this can be attached a rubber tube long enough to enable him to apply his mouth to the other end of it and inject food which he has masticated. In this way a collateral circulation is established. I will now introduce some milk into his stomach. You observe that when I withdraw the tube there is no regurgitation. The valve is a competent one.

The next patient is a case of cured popliteal aneurysm. The history is briefly that he is a man of fifty years of age; he has been in the Army, has had syphilis, and has not been a teetotaller. He began to notice pain and swelling at the back of his right knee in April, 1900. He came to the hospital on August 16th presenting a swelling which one had no difficulty in diagnosing as a popliteal aneurysm; all the typical features were well marked, there was a swelling in the course of the artery, with distensile pulsation arrested by pressure upon the main artery above; the swelling was compressible to a certain extent, and on releasing pressure upon the artery above, the swelling assumed its former size in the course of one or two heart-beats. He was not a particularly healthy subject, and the leg was the seat of old ulceration, pigmentation, and congestion; one was, therefore, a little afraid lest the leg should become gangrenous after ligature of the artery. His femoral artery was compressed for an hour a day for a week before the operation, and then the femoral artery was tied in Hunter's canal. Here is the scar of the operation. Everything went well for fourteen days; the circulation of the foot became re-established and the wound healed. At the end of a fortnight we thought it safe to let him get up for a short time. That turned out to be unwise, for it was followed by severe pain in the foot and the circulation became completely arrested, as far as we could see; the foot became anæsthetic, and for some days was on the verge of gangrene. But by rubbing and keeping the part warm the circulation in the foot became restored. But there is a curious point, namely, that the big toe has remained more or less anæsthetic ever since. He can now feel touch a little. I do not know what that anæsthesia was due to, and I do not know what the obstruction to the circulation was caused by, but possibly by some extension of clot from the original aneurysm, perhaps some embolism. He has had slight adhesions around the ankle-joint, but those have been broken down under an anæsthetic. If you examine the popliteal space you will feel the remains of the aneurysm. There is a small firm lump to be felt, there is no pulsation, it is not compressible, and it is obviously a fibrous mass left in the position of the original aneurysm.

The next case affords a very good illustration of the difficulty that sometimes arises in diagnosing between inflammatory and malignant disease. He came to the hospital two or three years ago complaining of aphonia, and on examining with the laryngoscope I found a swelling above the left vocal cord. I showed the case at the Laryngological Society, and opinion there was divided between syphilis, tubercle, and malignant disease—a difference of opinion which often exists. The case then came under the care of one of my colleagues at the hospital. He opened the larynx and removed a portion of the swelling for microscopical examination, but unfortunately that piece seems to have been mislaid. So when the patient came to see me a year afterwards, in the absence of my colleague who had operated, I was in complete ignorance of the result of the first operation He then had a swelling which was involving the outer aspect of his larynx, and I thought he had a recurrence of the malignant disease, which had got beyond the boundaries of the larynx itself, and so infiltrated the tissues outside. Thinking it might possibly be specific, I advised that he should be put upon iodide of potassium for some time before any question of a second operation was entertained. It seems that by some misunderstanding he did not have the iodide of potassium, but I was not aware of the omission at the time. As no improvement ensued I decided to operate. On opening the larynx I found that the left half of the thyroid cartilage was infiltrated with a swelling which extended to the muscles on the outer side of the cartilage. I was uncertain as to the nature of the swelling, and thought it would probably turn out to be malignant. However, as there was a doubt as to the nature of the case, and as efficient removal of such a large mass of malignant disease would be a very serious operation, I thought it would be better

* On June 20th he weighed 9 st. 6 lbs., and on June 30th 10 st

Digitized by Google

to remove a piece and wait for the result of microscopical examination. In due course the pathologist reported concerning the swelling. It then transpired that he had not been taking the iodide. When this omission was rectified the complexion of the case at once altered; the swelling inside and outside the larynx rapidly diminished, and in a few weeks completely disappeared. The left cord is quite fixed, but the laryngeal image is otherwise normal. His voice is harsh and rough, but not aphonic.

The last case I have time to show to-day is one of disease of the temporal bone, causing complete facial paralysis. I have cured the ear disease by the operation of mastoidectomy followed by skin grafting. The facial paralysis is much better, but, as you see, has not yet completely disappeared.

Gersuny's Subcutaneous Paraffin Protheses. L. Moszkowicz.—Gersuny has now an experience of thirty cases treated by the subcutaneous injection of paraffin, and the results have been invariably satisfactory. Two years have elapsed since his first experiments, and the result now is the same as at first, demonstrating that the prothesis can be considered permanent. The paraffin evidently becomes encapsulated in time, and persists indefinitely without change. The patient first treated—a prothesis of the testis, May, 1899, after bilateral castration—has since passed through a typhoid fever with temperature of 40° C. The paraffin seemed to be temporarily much softer at this time. Otherwise the artificial testes are the same as at first. No. 2 was a woman cured of incontinence of urine due to total loss of the sphincter and urethra. The paraffin was injected around the vesical sphincter and the ring pushed into the bladder. This forms a valve projecting inward, which has put an end to the oozing of urine, which had been continuous for five years. The effect has been permanently satisfactory. Pfannenstiel failed in a similar case as he did not reduce the paraffin ring, and consequently the valve opened outward, and was not water-tight. He injected paraffin with a melting point at 45° C., which is much too high, as it cannot be injected unless it is hot, and this favours its absorption and is liable to cause pulmonary embolism, which, in fact, did occur in his case. In patients Nos. 3 and 4, paraffin was injected into the palate or roof of the mouth to close a defect interfering with speech. Nos. 5 and 6 were injected in the anal sphincter to close a cicatricial defect in the sphincter left from a periproctitic abscess or extirpation of a rectal carcinoma. Prolapse of the rectal mucosa was cured in one patient by injecting 10 c.c. under the prolapsed tissue.—*Journ. Amer. Med. Assoc.*, July 27th, 1901.

THE PRACTICE OF BLOOD-LETTING.

By J. F. BRISCOE, M.R.C.S.

(Concluded from p. 176.)

THE following table will give the reader to some extent the practice of blood-letting in the eleven medical schools of London. A carefully recorded list of cases, collectively chronicled, might, in this generation of frequently recurring novelties in medicine, help a few of us in believing that a useful therapeutic agent has been cast aside with mistaken indifference. But as Professor Georges Hayem (Paris) says, "Statistics in blood-letting are scarcely utilisable, for it is very difficult to group facts which are often of desperate character in a satisfactory manner." The table which I have compiled from the eleven London medical school hospitals will speak for itself. Also the special table which Dr. Pye-Smith has kindly put at my disposal, of returns on venesection in Guy's Hospital. The same authority in the fifty cases which he brought before the Royal Medical and Chirurgical Society in 1891, under the heading of "The Therapeutic Value of Venesection," will, I believe, fully endorse the sentiments expressed in this paper.

The following two questions were put to the house physicians of the eleven London medical schools:

1. How often is venesection performed in your hospital?

2. Is there any special affection for which it is performed?

Digitized by Google

Hospital.	How often is venesection performed?	Special affections for which it is performed.
Guy's . . .	Occasionally	Right-sided engorgement of heart; chronic bronchitis or asphyxial conditions. Usually with the result hoped for.
King's . . .	Whenever it is considered desirable	See article by Dr. Duffin, 'King's College Reports,' vol. i, 1895. N.B.—This article strongly supports blood-letting, and points out useful and guiding lessons.
University . .	About three times in eighteen months	Cases of extreme overloading of right heart; renal inadequacy; for puerperal eclampsia and diphtheritic suppression of urine. In no cases of pneumonia does Dr. Batty-Shaw remember seeing it done.
St. Thomas's* .	Occasionally, but not as a routine measure	Uræmic convulsions; pneumonia; ingravescent apoplexy; urgent pressure symptoms due to aneurysm, bronchitis, etc. Venesection and lavage of blood has also been occasionally used in diabetes, typhoid, and pneumonia.
London . . .	Rarely performed	
Westminster . .	About twelve times in three or four years	For acute bronchitis with much cyanosis; uræmia; diabetic coma and apoplexy. Cardiac failure with right-sided engorgement, although we generally use leeches or wet cups for this condition.
St. George's . .	Not often undertaken	Heart disease, and much more rarely in cases of fractured ribs.
Middlesex . .	Very seldom	Mitral disease with embarrassed heart.
St. Mary's . .	It is done fairly frequently	In cases of heart failure, in mitral disease, in capillary bronchitis with cyanosis and dilated right heart, and, generally speaking, in cases when the right heart is labouring against an obstructed circulation, and showing signs of flagging; in aneurysmal stridor and sometimes in uræmic convulsions.
Charing Cross .	Not often—about four times in last eighteen months	Mitral stenosis with much failure and cyanosis.
St. Bartholomew's .	Venesection has been performed three times in one ward during the last six months, each time for pneumonia, once as a last resort in a case which suddenly became bad and ended fatally, once with decided improvement in a moderately severe case, and once with very speedy satisfactory results in a severe case. Sir Dyce Duckworth is the most faithful exponent of this method of treatment, and I do not think any student of this hospital looks upon the remedy as one in any degree out of fashion. (P. W. Rowlands, House Physician to Sir Dyce Duckworth.)	I have seen this treatment used in a case of uræmia without obvious benefit; in the status epilepticus with relief; and in a rapidly fatal case of cerebral hæmorrhage without change in symptoms. In severe bronchitis with failing heart we recognise its value. At a children's hospital I saw a child of eleven, dying of dilated heart in mitral insufficiency, regain compensation. (P. W. Rowlands, House Physician to Sir Dyce Duckworth.)

* Dr. C. R. Box, of St. Thomas's Hospital, informs me that he has on two occasions seen most extreme cases of uræmic convulsions so far recover after venesection as to be discharged from hospital. He has also several times found the same practice relieve in a marked manner the paroxysmal dyspnœa of intra-thoracic aneurysm when everything else has failed.

Digitized by Google

VENESECTION IN GUY'S HOSPITAL.

Year.	Sex and age.	Disease.	Ultimate result.*
1892	M., 42	Aneurysm of aorta; dyspnœa	Death.
	F., 42	Fatty and dilated heart	Death.
	M., 52	Granular kidney; large heart; bronchitis	Relieved.
	F., 24	Atheroma of pulmonary artery; failing heart	Death.
1893	M., 47	Bronchitis with emphysema	Relieved.
	M., 48	Bronchitis with emphysema	Relieved.
	M., 36	Pneumococcal empyema	Death.
	M., 52	Cerebral hæmorrhage; granular kidney	Death.
	M., 33	Uræmia	Relieved.
1896	M., 52	Bronchitis	Relieved.
	F., 45	Bronchitis	Death.
	M., 52	Bronchitis	Relieved.
1897	M., 49	Bronchitis with emphysema	Relieved.
	M., 32	Aneurysm of aorta	Relieved.
	M., 47	Aneurysm of aorta	Death.

* Temporary result, always more or less relief.

These fifteen cases during four years were carefully compiled by Dr. Pye-Smith and the Medical Registrar of Guy's Hospital. The results appear to be satisfactory. Dr. Pye-Smith remarks, "Bronchitis with great cyanosis, aneurysm, and uræmia, are the best cases for bleeding. I have several times bled in pneumonia and in valvular disease of the heart to relieve great dyspnœa and cyanosis, but I have never seen such striking relief in these cases as in those above mentioned. On the other hand, I have never seen bad results." However, as Dr. Duffin says, "we owe a deep debt of gratitude to Sir Samuel Wilks for the light he has thrown on this difficult subject." Among others who have been generous enough to give me their valuable opinions on the subject, I have to thank Dr. J. W. Ogle, whose Harveian oration in 1880 I have had the pleasure of perusing, and whose courteousness has been unbounded. Not least have I to mention the name of Sir Dyce Duckworth, who is one of the most faithful exponents of blood-letting, and to whom I am much indebted. This authority reminds us in a paper "On the Modern Neglect of Leeching in Practice," 'Liverpool Medical and Chirurgical Journal,' Jan., 1897, that "to imagine for a moment that we of to-day are alone in possession of the best methods of therapeusis ever known in the history of medicine, is to suppose that there never were any great physicians before the end of this century—*quod est absurdum.*" With regard to references, beyond the few I have mentioned there is a concise and clear monograph on the abstraction of blood by Dr. Duffin, vol. i, 1895, 'King's College Hospital Reports.' In Table 133, 'Epitome of Current Medical Literature,' Thirteenth International Congress of Medicine, 'British Medical Journal,' September 29th, 1900, "The Indications for Bleeding," by Professor Georges Hayem (Paris), is briefly yet clearly summarised, and is entirely in accord with the views and dictation of those who practise blood-letting in London. To those who know the writings on blood-letting of the older school of thought, whose teachings have been handed down to us, will find in this discussion at the Congress nothing beyond what was taught and laid down by Dr. Markham, a previous editor of the 'British Medical Journal,' Physician to St. Mary's Hospital, and the Gulstonian Lecturer for 1864. So that, although modern clinicians are alive to the therapeutic value of venesection, we owe the wave of its revival in London to the conservative policy of those physicians whom I have named in this paper. These are scholars and teachers of mature years, and have always been favourable towards its proper practice. The original and powerful thinker, Professor Cleland, of Glasgow University, at a meeting of the Glasgow Medical Society several years ago, stated "that it was time to consider how far blood-letting might affect the constitution in other ways than by depletion." In a paper by the same authority, "Certain Aspects of Corpuscular Action," read before the Glasgow Medical Society, January 22nd, 1892, he states, "At that time (referring to a previous paper) I pointed out that it was probable that young red corpuscles were more likely than old to be efficient as oxygen carriers in the blood, and that one of the effects of blood-letting was to favour the production of new corpuscles. But what I said then with reference to red corpuscles is probably more important to be recognised with reference to leucocytes. The liberation of these from all the sources in which they are developed would appear to take place rapidly after loss of blood."

So many have kindly given me assistance in preparing my paper that, if I were to enumerate all, I might appear to be unnecessarily gushing. Yet I feel indebted to them, and this slight acknowledg-

Digitized by Google

ment is all I can offer. For the full literature on the subject Dr. Neal's 'Medical Digest,' a complete "enquire within" upon everything in medicine and surgery, will supply the wants of everyone from the evidence there recorded of vicarious hæmorrhages, the self-bleeding rhinoceros, the magic of blood-letting in cattle diseases, to the practice of cupping by the natives of Africa. While concluding my paper, and expecting returns from the north of England, Scotland, Ireland, and elsewhere, I have received from Dr. H. C. Drury, of Dublin, the particulars as to the practice of blood-letting in Ireland. This physician, who is attached to two of the most representative hospitals of Dublin—Sir Patrick Dun's Hospital and the Fever Hospital—writes as follows:—"Only once in my recollection, about twelve years ago, in a case of engorged right heart during pneumonia, have I seen venesection performed in my two hospitals. The external jugular vein was opened with immediate relief, but death occurred afterwards." The same authority says, "I have no accurate information, but I believe the practice of blood-letting has been practically given up in Ireland, even by the older practitioners, and this refers also to wet-cupping." However, Dr. H. C. Drury remarks that in Ireland dry-cupping is frequently resorted to with apparent success in pneumonia where there is considerable accompanying bronchial catarrh throughout the lungs, and also in acute nephritis with very scanty urine.

To consider the question of blood-letting in kidney affections, it is well to discuss the anatomical relationship of the vascular area of the loin; and this anatomical question, of the direct removal of blood from the kidney substance by cupping over the loins, is not, as far as I am aware, clearly grasped by all students of medicine. The renal arteries give off small branches to the supra-renal capsules, the ureter, and the surrounding cellular membrane and muscles; these anastomose with the abdominal branches of the lumbar arteries. There is, moreover, a circle of anastomosis between the lumbar arteries, with the epigastric and internal mammary in front, the intercostals above, and those of the ileo-lumbar and circumflex iliac below. But there are two other points to consider, namely,—(*a*) the action on cupping on the vaso-motor nerves, and thereby acting on blood-vessels at a distance; (*b*) the local action of the blood contained in the muscles and tissues in the region of the loin. This will explain to some extent the statement by Dr. Duffin, "that the communication between the loin and the kidney is even more direct than the connection between the lumbar arteries and the renal artery." It is more direct, Cohnheim and D. M. Litten would say, since they point out that there is a communication between the glomerular element of the kidney and the loin through the capsule. This, then, is how they explain the suction action of the cupping glass to which they refer in their works on embolism. But in attempting to thrust upon the student of the practice of medicine and surgery the dictation of a former school of thought which has not been barren, I will remark that the teaching of those now in the autumn of their lives is not to be thrown aside by the modern student. For instance, in regard to cupping I find I have made a grievous error in my article on "Conditions Indicating Blood-letting," *vide* CLINICAL JOURNAL, September 20th, 1899, and in which I state, "but it is clear to you that no direct abstraction of blood from the kidneys on anatomical principles would occur from topical blood-letting." This error, for which I apologise, is clearly and distinctly opposed by William Turner, Senior Demonstrator of Anatomy, University of Edinburgh, in an article in the 'British and Foreign Medical and Chirurgical Review' for the year 1863, on "The Existence of a System of Anastomosing Arteries between and connecting the Visceral and Parietal Branches of the Abdominal Aorta." The same anatomist, now Sir W. Turner, has another article in the same journal for 1865 on "A Supplementary System of Nutrient Arteries for the Lungs." But this journal has ceased to exist since 1877, and I know of no journal in the United Kingdom out of the seventy or more of the present day which takes its place, or fulfils the same scope. The anastomosis of the abdominal vessels is clearly described by Sir W. Turner in his article, and it is the outcome of a series of injections, some of which are preserved in the museum of the University of Edinburgh. These injections were proved and tested, so that the injecting fluid could be made to pass from kidney to intestines, to the intercellular tissue and to the vessels of the abdominal walls, and *vice versâ*. This subperitoneal arterial plexus serves as a sort of safety-valve, and diminishes the tendency to extravasation and effusion

Digitized by Google

into the kidney and other organs. Sir W. Turner says, "Thus, perforative arteries pierce the fibrous coat of the kidney, and would appear, not only to serve the highly important office of connecting the renal trunk with the arteries of the abdominal parietes, but, in addition, they doubtless fulfil a functional duty of some moment. The argument, then, in favour of local blood-letting in the loins has thus been proved by the vascular communication between the integuments of the loins and the kidney. So there is good support for applied therapeutics to the lumbar region in renal cases." Not only is this so with the abdominal cavity, but it also applies to the thoracic walls, for, as Sir W. Turner points out in his second article above mentioned, there is also in the thorax a sub-pleural mediastinal arterial plexus divided into anterior and posterior. This plexus forms the supplementary system of nutrient arteries for the lungs, and the bronchial arteries are not the only nutrient vessels of these organs. The plexus thus affords a medium of communication between the visceral and parietal arteries, and in the thoracic cavity there is an arrangement closely comparable to that of the sub-peritoneal plexus in the cavity of the abdomen. Mr. Jacobson pointed out to me that the credit of describing these plexuses is in this country rightly given to Sir W. Turner. Quain, in the last edition, the best English work we have, quotes from Sir W. Turner's paper as to both plexuses.

The information I have received from Dr. John F. Christie of Aberdeen is to the effect that dry-cupping is occasionally performed in Aberdeen for œdema of the lungs. But with regard to the practice of venesection in the Aberdeen Royal Infirmary it is seldom performed. That is to say, in the last thirty years it has been practised about three times, and that for cardiac cyanosis. Dry and wet cupping is taught in the Royal Infirmary, but it has not been practised for some time.

In approaching the concluding paragraph of this paper, I should like to cursorily mention the controversial aspect of past and present methods of Research in Medicine and Surgery. If we believe that the older school of practice was wrong, we yet ought to conceive that their practices have not been completely barren. If we do not hold this truth of them, what will the next generation of thinkers say of us? The fact is, the new bacteriology, the new psychology, and the new surgery are indirectly the fruit of the work of older heads, and we have yet to prove that our successes are to equal them and extend beyond our anticipations. Are we not indebted to the experience of a past generation, and will not the next benefit from this period, and so on *ad infinitum?* To expunge old remedies from our catalogue of therapeutic agents is a fashion of the present age. The cyclical course of events may cause us to retrace our steps, for, as Sir Dyce Duckworth says, "In adopting any new method of treatment, we have to keep our heads, to weigh most carefully the merits of such practice, and compare it honestly with the ripe experience and wise teachings that have come down to us from those who have preceded us, remembering that 'our forefathers were not all fools,' and that the full light of mid-day is not perhaps yet shining upon us even at the close of the nineteenth century." However, we must acknowledge that our whole knowledge of the blood and of living corpuscles is changed since the old days of venesection. The old people knew little about the leucocytes or colourless corpuscles of the blood. The existence of amœboid corpuscles or wandering cells was first pointed out by Recklinghausen in Virchow's 'Archives,' vol. 28, 1863. Then it was at a later period, as Professor Cleland points out in his 'Human Anatomy,' p. 2, after Wallis's discovery of diapedesis had been reflected for years, that it was rediscovered by Cohnheim, who pointed out its great pathological importance. Now we know that the leucocytes have their origin in the solid tissues, and, many of them, return there.

Summary of Blood-letting.

1. Careful incision; open the vein obliquely; shampoo limb if blood does not flow, and apply the same to the other extremities; keep warmth to the body; to encourage the flow of blood give an inhalation of nitrite of amyl, either with wet-cupping or venesection pure and simple; after operation keep patient quiet in the recumbent posture and in a darkened chamber; administer milk and diluent drinks, and relieve the intestines with some suitable aperient.

2. In certain cases of heart disease, uncomplicated mitral regurgitation when compensation has become inadequate, there arises a gorged and distended right heart and lung with occurring hæmoptysis. Six to twenty or more ounces may be re-

Digitized by Google

moved in these cases. After the operation, drugs like digitalis and diuretics, which have previously failed, will complete the relief.

3. Sudden deprivation of pure atmospheric air from a healthy lung causes the right heart to become distended and paralysed. This chemical origin of cyanosis is shown in bronchitis and in all gaseous suffocations, and the practice of blood-letting in these cases must be bold and thoughtfully considered. Venesection is thus the therapeutic agent.

4. To discriminate between two conditions :—

(*a*) When a patient is much choked, labouring hard with his breath, and moreover with his veins distended, the heart is dilated, then bleeding is our sheet-anchor. It is obvious with this state we get a small pulse, and when the distended right heart is emptied the pulse becomes full.

(*b*) On the other hand, the patient is choked with bronchitis, but his right heart is acting well. His surface wears a leaden hue from the free circulation of venous blood in his arteries. The pulse is good but of high tension from the quality of the blood contained in the arteries. The venous system and the liver are not engorged. Bleeding is not indicated in this class of case, and is an instance of one of the errors and abuses of blood-letting. The right heart is quite able to take care of itself so far. As Dr. Hare 'Good Remedies out of Fashion,' puts it, the right remedy in this class of case is the "emetic." He says, "In suffocative bronchitis the effect of emetics is sometimes magical, and by their administration in such cases not only is immense relief given, but I verily believe—I am certain—that lives are saved." This, then, with respiratory stimulants is the proper treatment when the bronchial tubes are choked.

5. Avoid bleeding weakly people, broken-down old men, and drunkards. Venesection is also a doubtful agent in acute peritonitis, but a few leeches may give relief.

6. In capillary bronchitis and in pneumonia venesection may save lives. In the former, where there are signs of distension of right heart; in the latter, when there is a large extent of lung involved with symptoms of cyanosis and heart paralysis. In pleurisy of a diaphragmatic nature venesection is indicated. In pericarditis and pneumothorax; bleeding in the former is useful when orthopnœa, irregular pulse, distended jugulars, and arterial anæmia are present, should these signs not depend upon excessive effusion into the chest. In the latter when there is arrest of circulation in the collapsed portion. Again, in thoracic aneurysm and tumours of the mediastinum venesection is advisable, as it relieves painful tension and assists the action of iodide of potash. Only a few ounces should be allowed to flow so as not to impoverish the blood.

7. In acute nephritis and uræmia blood-letting is sound practice.

8. In lumbago and certain forms of headache of a congestive nature. Dry cupping will be useful in the former, and bleeding, either from the nose, by leeches, or venesection, will give the necessary relief in the latter.

9. When symptoms of impeded venous flow are present in cranial affections, indicated by laboured breathing, surface veins full, and face congested, venesection is to be practised.

10. Blood-letting should not be practised in a routine fashion.

The above summary will give us some idea of the principles and practice of blood-letting. The practice, as will be seen, requires an intimate acquaintance with disease, the processes of disintegration of the body, and a thorough knowledge of the laws and functions of the human machine. So that the ignorant practitioner and the charlatan are alone responsible for its abuse. Its action is for the relief of tension, distension, and the removal of effete chemical matter. Its therapeutic value is direct, differing from many agents which act only indirectly.

There is a growing tendency in these days to look upon the practice of surgery and medicine as a business or trade. True business qualifications are essential for every professional medical man, but there is another debt we owe to society, which they do not all seem to grasp, namely, the science of our calling, which is not a thing to be carelessly talked about. The fundamental principles of science is truth, and from this arises the true growth of our profession. It is our duty to know why we administer our drugs, antitoxins, and other therapeutic remedies, not to thoughtlessly apply them because we are told they are beneficial, but to work out our own scientific salvation.

In closing the material of my text, I will ask you not to be too fashionable in your practice of medicine and surgery, but to listen to the dictation of those whom I have quoted in this paper. Take the lancet from your pocket, and you may save the life of your patient, as many have done before you.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY

No. 462. WEDNESDAY, SEPTEMBER 4, 1901. Vol. XVIII. No. 20.

CONTENTS.

PAGE

*A Clinical Lecture on Raynaud's Phenomena. By JONATHAN HUTCHINSON, F.R.C.S., F.R.S. 305

*With Mr. Stansfield Collier at the Hospital for Sick Children 315

*Two Cases of Swelling of the Eyelid occurring after a Bath. By THEODORE FISHER, M.D., M.R.C.P. 318

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A CLINICAL LECTURE
ON
RAYNAUD'S PHENOMENA.

Delivered at the London Hospital, June 19th, 1901,
By JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

GENTLEMEN,—It is always pleasant to an old man to renew the avocations and pleasures of his youth, and so I come before you with a subject which reminds me of my days at the London Hospital and of the work in the wards. I believe that some of the earliest cases of what is known as Raynaud's disease in England, not in Europe, which were observed and studied under the name of Raynaud's disease were studied in the wards of the London Hospital. So it is a special pleasure to me, as I always select those subjects which I have had previously under study in the wards here, to bring before you to-day this important and very interesting disease.

And first I would say I think we shall all gain a clearer idea of our subject if we decline to call it Raynaud's disease. The expression Raynaud's disease would imply that there is some one malady, complete in itself, having all the symptoms the same in all cases. Now that is not the case; there is no one malady which we designate by the term Raynaud's disease. We know what we mean by syphilis; we know what we mean by variola; we mean a sum total of symptoms which is the same in all cases; the same may be said of pulmonary phthisis, we know what we mean there. But in the case under consideration there is no one disease which is the same in its cause, which is the same in its phenomena, the same in its final results in all instances, which deserves the name of Raynaud's disease. Please understand that I wish to do all honour to such a man as Raynaud—a man of singular originality of mind, of great pertinacity in observation and clearness of thought, a man who worked sincerely and devotedly in the pursuit of clinical truth. I would do all honour to the name of such a man. He enriched the science of medicine by his observations, and if we could

Digitized by Google

do honour to him more, or if we could stimulate others in the future to emulate him in their modes of research by so doing, let us call it Raynaud's disease for ever. But I think we shall do quite enough honour to him if we pursue the methods that he did – *i. e.* attempt to be precise in the signification of the names which we employ, and not use them loosely. And if we speak rather of Raynaud's phenomena than of Raynaud's disease I think we shall be on safe ground, for Raynaud's phenomena are things which we understand, and which are the same in all cases. There is no such thing as Raynaud's disease, and under Raynaud's phenomena we shall have to include a large group of maladies which are very different as to their causes.

What are the phenomena which I propose we should know as Raynaud's phenomena? You will probably be aware that expressions have been used, such as local syncope, local asphyxia, symmetrical gangrene of the extremities, and these have been used as synonymous terms; recently the prefix acro- has been applied, and I think it is a very useful term to employ in that connection. Under these different terms we imply a knowledge of the circulation in the extremities, viewing them as extremities, because they are at the extreme periphery of the circulation, and liable to be interfered with, and that same interference with the circulation may be an interference with the nutrition of the parts, and lead to local symmetrical gangrene, which is one of the terms used to denote this condition of the extremities. I think, then, we had better begin by simplifying our problem, and therefore recognise that we are all subjects of Raynaud's phenomena in a greater or less degree, that we are all liable to have our hands made very cold, we are liable to have them become cold, and not only cold through exposure, but through the influence of the nervous system. I daresay all of you are familiar with the fact that the circulation in the extremities may be interfered with, not by mere external temperature—everyone is liable to that—but through the influence of the nervous system. Our next assertion will be that no two of us are alike, that we are all different as regards the susceptibility of our tissues and our circulation to disturbance from separate influences. If we are all out on a frosty day, we should not each manifest the same conditions of circulation in the tip of the nose, the hands, and the ears. One would be warm, another cold, and another would experience no great inconvenience. Here, then, we recognise at once that there is such a thing as personal peculiarity, which is an extremely important thing to realise. Each person is born with a personal peculiarity, which will render him liable in greater or less degree to disturbances of this kind. The term local syncope is given to the phenomenon which is sometimes known as "dead finger." Some persons on putting their hands into cold water will be liable to have them become dusky and dead. All of us, if we are unduly exposed to cold, will be liable to have the hands become either very dusky or very pale, or both pale and dusky, or pale and mottled at the same time. And some persons will be liable to have one finger, or all the fingers, become absolutely dead and white. When the finger is taken out of the water the circulation of that finger may be seen to be quite suspended. To that condition the term *digitus mortimus* is given, and that condition depends upon a spasm of the arteries supplying the finger, a cutting off of the blood which the finger should have brought to it. This we can easily realise. That is the condition which is known as local syncope, the absence of blood in the part which becomes pale. Local asphyxia is the condition in which the part becomes not pale but very dusky, and of a livid hue though not shrivelled. In local syncope the part would be shrivelled; it would be like the finger of a corpse; the end of the finger, which should be plump, will be shrivelled up, just as the fingers of a corpse would be, owing to the absence of fluids in the part. So we use two terms, local syncope and local asphyxia. We say that local asphyxia is due to fulness of the small veins of the part, whereas local syncope is due to the absence of blood in the veins and arteries. In both conditions the arteries are more or less affected by being thrown into spasm. In the condition of local asphyxia, as contrasted with local syncope, the closure of the lumen is less complete. That is a thing which we can very easily infer must be the case.

Is it true that the conditions of local syncope and local asphyxia always or usually go together? Perhaps they do, or perhaps one precedes the other usually. Perhaps the first stage is oftenest that in which the extremity becomes very pale and then

Digitized by Google

livid. But I do not think we shall aid ourselves very much in our perception of this process by insisting too strongly upon these phenomena having any relation in time to each other. In some persons the fingers become dusky straight off, in others they become pale before they become dusky, and in some they become pale and remain so throughout. In a great many cases there is a certain amount of dusky mottling and pallor mixed up together, as in a corpse. Such, then, are the uses of these terms.

Next, I have to ask your attention to the fact that in order to constitute a typical resemblance to Raynaud's phenomena the patient ought to be liable to have the phenomena in a paroxysmal manner, that is to say the symptoms ought to come and go. The periods of liability may in some cases be prolonged, that is to say extending over several weeks, and a patient may be liable to a prolonged period during which there are certain variations; but during the greater part of the time the limbs remain in a state of extreme duskiness or of extreme pallor, one or other of the conditions varying a little. And the phenomena may recur after a long time. Still, that it should be paroxysmal is of the essence of the disease which we associate with the name of Raynaud. In saying that I am quite prepared to admit that there are cases which are not paroxysmal, which occur only once in the patient's life, and then pass away and do not come again; and these cases we must consider side by side with the others, although they are not exactly parallel.

I might just deal with the question as to whether a symmetrical gangrene of the extremities should rank as being precisely synonymous with Raynaud's phenomena or Raynaud's disease. I will relate two cases which came partly under my own observation; the subjects were healthy men. In one case the patient was a policeman in good health, as far as he knew, who filled his ordinary avocation in every way. The other man was a paper-stainer. The two cases were quite alike in this, that the trouble involved them in the loss of all the ends of their fingers, the tip of the nose, and the ends of the toes. Drawings of the cases would lead one to suppose that they had got a black kid leather glove drawn over the fingers. The fingers were perfectly black and mummified, and in each case the ends of the fingers sloughed off, and the ends of the toes and the tip of the nose and the tips of the ears sloughed away. And then the men got quite well. Now, in neither of those cases has the patient been liable to any disease subsequently. Both of them had been carefully examined as to whether they were the subjects of renal disease, or whether the heart or anything else was wrong, or whether there was anything which would be capable of accounting for this attack of local gangrene; but nothing of the sort could be found in either of them. In neither of them was there anything which had occurred in the height of the attack which would explain the symptoms; neither had been exposed to unusual cold or fatigue. But in both the history of the case was that suddenly the extremities became painful, the limbs began to ache, then the aching was extreme, and that then the patient was placed under medical care and was put to bed, where he went through a severe illness, so severe that it was thought he might die; and that illness was attended by symmetrical gangrene of the extremities. I think these are instances in which there is no evidence of susceptibility of the nervous system. A patient may be liable to asphyxiated extremities, because that part of his nervous system which is concerned in regulating the size of the arteries by the vaso-motor mechanism is very susceptible. If my hand on being put on to a cold substance has an impulse of cold transmitted to the nerve centres and throws my arteries into a condition of spasm, which is quite reasonable to suppose, then by the degree of irritability or susceptibility to that should be measured the tendency to local syncope and asphyxia, and that will vary in different individuals within the bounds of health. If it goes to an extent which is morbid, so that the slightest exposure to cold—as is the case with some people—will at once induce either a reflex dilatation of the veins or a reflex contraction of the smaller arteries, then the term which we are now discussing becomes applicable. That there are people in whom this does occur, passing gradually from its normal physiological condition into disease, we are all ready to recognise.

As regards the two cases I have mentioned, in neither of them was there the slightest evidence of irritability of the nerve system. Both of them were strong, robust men; not nervous men, not men

Digitized by Google

in whom the capillaries were liable to dilate suddenly, not men accustomed to blush on slight provocation; they were two healthy labouring men in middle life, and it is impossible to suppose that the vaso-motor nervous system in its ordinary methods of reflex disturbance, was in any particular abnormal in them.

What was the nature of the illness then? Both of them were in bed for a considerable time, and as I have said, were very ill. I did not see them during their illness, nor have I any accurate details as to the nature of the illness. Their illness was certainly regarded as obscure, and it was attended by sloughing of the extremities, as I have mentioned.

Now, in endeavouring to interpret the phenomena of Raynaud's malady, I will ask you to keep in mind three or four different elements which may go into partnership in a case, one of which may greatly preponderate over the others, but all of which probably take shares in the case. First we think of the vital endowments of the tissues themselves. In none of us, in no two individuals, are the vital endowments of the solid tissues themselves exactly the same. Next I will ask you to have regard to the arteries themselves, the arteries being the means of conduction of the blood to the peripheral parts. Those arteries may be diseased or they may be sound; they may be very small or they may be very large; they may be very liable to be thrown into muscular spasm or they may not. Perhaps there I am trenching on my next point, which is, that we take into consideration the state of the nervous system, whether the reflex irritability of the muscular part of the arteries is morbidly irritable; and whether the nerve centres which regulate the amount of the blood-supply are in a state of irritability, so that a slight cause will induce them to throw upon the arteries an undue amount of influence, causing vaso-motor contraction.

I have dealt with the tissues, then with the arteries, next with the nervous system, and now we have to consider the central organ of the circulation itself. The heart itself may be weak, it may be very weak; and if the heart were so weak that it could only just propel the blood to the extremities, just as in a case of general syncope, in which the patient faints, and for the time being ceases its action; if it were possible to depress the heart's action to that point at which it could only just carry on the circulation and keep the patient alive, we might explain the condition in which the blood could not get to the very ends of the fingers, the ends of the toes, or the tip of the nose. And manifestly those parts are at a disadvantage in being at a distance from the heart, and we should expect to see here the results of any remarkable failure in the heart's power. That is the hypothesis which I suggest as a probable one in the two cases which I have narrated; that very probably they were cases in which some unrecognised serious temporary condition occurred in the heart—an inflammation of the heart substance, or something which very much depressed the power of the heart, and that it was that which led to the gangrene. The patients have recovered; they are no longer liable to recurrent attacks of the trouble. I must exempt the nervous system; I must exempt the tissues; the fingers in both cases have healed well, and the circulation has now returned; the policeman has gone back to his duty, and in both cases the men are enjoying very good health.

These, then, illustrate the class of cases in which I would suggest that the heart itself is at fault—a suggestion which I make with some diffidence because I have not proved any heart disease; no one has found any murmur, no one has found that the heart was extremely weak—but I suggest it because I do not see any other hypothesis to explain it, unless the patients were liable to blood poisoning. And there, again, I think the way in which that would produce its effect would be by acting chiefly upon the central organ of the circulation.

I have adverted to these different elements which go into partnership in a case of Raynaud's phenomena. I am very much interested in asking students of medicine to engage widely in their pursuits, and not to be content with studying in the wards of the hospital, but to study also diseases of the lower animals; and even to go more widely and study the diseases of vegetables, and I believe you will get some very interesting work during your vacations in the country, and you will find very interesting subjects for observation among animals and plants. And further than that, you will not only find things which are interesting, but you will find in that way some light thrown upon problems of pathology in reference to the human

Digitized by Google

subject. I am sure the study of diseases of plants and animals is not so entirely apart in their bearing upon our special pursuits as some at first sight might be disposed to infer. You see we can study the phenomena of acro-asphyxia, and can see the point of death in plants under conditions in which we get rid altogether of the nervous system, for the simple reason that they have not got one; in which we get rid, so to speak, of the central organ of the circulation to some extent, but not wholly. Plants have no heart, it is true, but the plant has in reference to circulation what is equivalent to a heart in its roots. There is no question that the distribution of sap and its ascent into the tree, to its topmost boughs, is accomplished partly by the *vis a tergo*, that is to say, suction, so to speak, caused by the growth of the tissues of the growing bud, and partly it is accomplished by propulsion from below. And that is not by any heart, there is no contractile muscle or heart; but in the extreme end of the roots which are engaged in the soil below there is a power, a something driving this sap upwards, which must be considered as a power somewhat similar to that by which the heart drives blood to the fingers. That is a very different thing, but still it is a *vis a tergo*. There is a circulation in the plants and there must be circulation in every living thing, in plants as well as in animals, or the thing is dead. There must be circulation to keep the tissues living, or else those tissues will die; they must be renovated by a constant movement of fluids. So in attempting to explain the ascent of sap in vegetables in the spring we take into consideration the structure of the plant. Tubes are there; there are in the outer layers of the white wood little tubes through which, by capillary attraction or partly by propulsion, and partly by capillary attraction, and partly by exosmosis and endosmosis, the fluids pass. Now, to prove that the rootlets do drive the fluid up, I may mention that you may cut across a birch tree three feet from the ground, and leave not a single bud or leaf attached to it; and leave that standing through the winter. Wait till the spring comes. What will happen? The top of that tree, which has been perfectly dry during the whole of that winter, will begin to pour out fluid, and that fluid will run down it in large quantity. What is the force which drives that sap up? It is clear there must be a force originating below; there must be a root force which drives it up. So I assert there is a *vis a tergo* which is generated by the root below.

As to what lessons the diseases of plants and trees may give us in reference to our interpretation of Raynaud's phenomena, if you will look at almost any collection of trees or plants anywhere it is very easy to find examples. You will see many examples of acrosphacelus; the ends of the leaves are dead, that is to say, the very tips are dead, but the rest of the leaf is living. Or you may notice in a very high tree the very tops of the twigs are dead, or the branches have been unable to produce their leaves, and are comparatively bare, and if they are not absolutely dead they will be dead next year. Here, then, we have examples from very familiar cases in which the ends will fall off, the very extremities of the living tissues will die and fall off because they could not get nutriment enough, and the want of it is the reason, because it was at such a distance from the origin of the circulation or *vis a tergo*. Now, in the gangrene and death of the extremities of plants and trees, without the whole of the tree dying, we have examples indicating that the tissues and cells have been implicated; they may have been attacked by cold, and that cold may have been enough to kill the ends of the branches though it was not sufficient to kill the whole of the tree, only the more exposed parts or the extreme periphery of the organism. It is, of course, the peripheries which are most likely to be influenced by the cold of the surrounding atmosphere. And next we have to suggest that the tree, as in the case of the animal, is liable to be influenced by anything which enfeebles its roots; and if you see a tree is dead at the top but living below; or if it is dead at the ends of all its branches while it is still producing a few leaves near the trunk, you may be almost certain that that tree has something amiss with its roots, which are unable to send the sap to those parts which are deficient, just as I have supposed in the case of these two men who had the ends of their digits dead, that there was something in the central organ of the circulation which enfeebled it and reduced the *vis a tergo*, and that this diminution of *vis a tergo* produced gangrene. So I suggest as regards plants, that they are foremost instances in proof that the tissues themselves, or the peripheral parts of them, are more liable to be influenced by external cold, and that we can easily understand in

Digitized by Google

the human subject, and that they also show the want of vigour in the propulsion of the roots, equivalent to the heart in living animals, and that that want of vigour may be a cause of this death.

I might further say that they also illustrate disease of arteries, for if the little tubes of the wood tissue which carry the sap up become blocked, you may easily see that death of the part of the bud will ensue. So in calcareous arteries, if the arteries become calcareous, their calibre much diminished, the risk of acrosphacelus of the part below is obviously very much increased.

Now to be practical. I have mentioned two cases in adult men, not nervous men, not the subjects of any malady, who have become the subjects of symmetrical gangrene; and I have asked you to believe it is probable that they were the subjects of some acute heart disease, which I think is the most likely cause if the patient be capable of acting properly afterwards.

Now a few words about the more common forms of the malady in which we recognise Raynaud's phenomena. In young people we may speak of a juvenile form, which occurs not in young infants, for in a young infant the nervous system is not as yet perfectly developed; no very young infant ever becomes the subject of herpes zoster, which is a disease produced through the nervous system. Similarly no very young infant becomes the subject of any form of morphœa. Morphœa, again, especially the zoster-form morphœa, is a disease which is distributed by the nervous system. But children of five years of age and onwards may have herpes zoster, or they may have morphœa. But, as I say, we have to wait until the nervous system is well developed before any of these nervous system diseases come on. So young infants are not the subjects of Raynaud's malady, but children may be and not infrequently are.

I might have mentioned chilblains as one of the acrosphacelic conditions, conditions of disturbed nutrition, occurring upon the extremities, because those extremities are in peculiar states as regards circulation, and chilblains have an interesting relation to Raynaud's phenomena, but they are quite different from Raynaud's phenomena. In them there is always local inflammation, and a number of considerations have to be taken into view in order to explain them and their differences. In Raynaud's phenomena there is no inflammation, but suddenly the circulation is comparatively suspended. Children are more liable to chilblains than adult persons. Again, infants very rarely suffer from chilblains; a child must have arrived at a certain age before it has any liability to chilblains, because chilblains have a good deal to do with the nervous system. The typical cases in children are cases of extreme asphyxia—blue hands, blue cheeks. On a very cold day many children will be seen with very much congested cheeks, the cheeks becoming almost livid from the exposure. Typical cases of Raynaud's phenomena, which are more or less dangerous, sometimes running to gangrene, and in which the gangrene may extend from the digit to the whole of the hand or foot, involving sometimes the leg and sometimes the forearm, may be induced not by blood disorder, but through the influence of the nervous system.

I will suggest respecting the juvenile form of Raynaud's phenomena that it is liable to occur over and over again in children who become liable to it through the enfeebling influence of some illness, that in some way they have been liable to attacks of asphyxia, and in them the lividity is often extremely great and the limbs become swollen. In the extreme cases of asphyxia there is turbidity, evidence of turgescence, and other symptoms preceding gangrene. I believe this group of cases of Raynaud's phenomena always occurs in young persons.

I venture to describe another form, which occurs in old people, or at any rate those who have passed middle life, but not in the very old. In children I suggest that the nervous system takes a very large share. When we come to old people past middle life, if they become the subjects of Raynaud's phenomena you must listen to the heart and feel the pulse. Senile gangrene in the ordinary acceptation of the word, as that which occurs in the foot almost invariably in elderly people, must be thought of apart from Raynaud's phenomena, because it is scarcely ever symmetrical; it is very rare indeed that you see it occurring on both feet. What is our inference from that? It is that local causes and not the general influence of the nervous system or of the heart which have to do with senile gangrene; and I believe you may say it is an established fact that the senile gangrene which you see frequently in hospital wards in elderly people results from some

Digitized by Google

slight injury to an extremity in a person in whom the arteries are diseased, calcified, calcareous structures, arteries whose lumen is diminished by calcification, that is, a condition which renders the foot in an elderly person gangrenous from an injury which in a younger person would simply have inflamed or would have healed up without inflaming. Senile gangrene is related to some forms of Raynaud's phenomena, but as it is not symmetrical we shall still have to use the term senile gangrene, as our forefathers did, for a malady which does not manifest itself on both sides, and is not usually paroxysmal in character. If ever you see a case in which both extremities are affected, and in which the two hands in an old person are affected as well as the feet, you must investigate it on the lines on which you would investigate another case of Raynaud's phenomena, because it shares in the phenomena of Raynaud. I was in Great Yarmouth one day, and as I had a few hours to spare I went into the German Hospital there. The house-surgeon there kindly showed me the cases. He showed me one old man, of eighty years of age, who had the ends of all his fingers in a state of gangrene. Now, in that man the arteries were so calcareous that I could scarcely feel the pulsation. The affection was in his hands and not in his feet, affording an illustration of what I have been saying. This old man had been in the hospital several years before for what was then accounted the ordinary form of senile gangrene, that is to say, affecting the great toe of one foot only. He was an old man who enjoyed good health; he was not nervous, and there seemed to be no nervous element in his case, and we had to ask what influence had been brought to bear upon him. I have said his arteries were calcified and very small, and he had gone about for years with a circulation at a great disadvantage owing to progressive senile calcification of his arteries, which were getting blocked up. What was it that had induced the symmetrical attack in both hands and yet had left his feet quite free? There was the problem. That I am at a loss somewhat to say, but it occurred quite suddenly, and not in connection with any special exposure to cold; exposure to cold would not tend to influence the hands more than the feet, because a man can easily protect his hands in various ways more than the feet. He was a man who was not exposed unduly; he was living in his cottage and had nothing particular to do; but one day he suddenly found the ends of his fingers quite black, and he lost the ends of them all. In such a condition we put aside the nerves, and I think that is fair. There is no doubt the influence of the nervous system had nothing whatever to do with it. There was no obvious disease of his heart; but still I am inclined to think that the explanation of the sudden occurrence of this in his hands was not only predisposed to but almost brought about by the calcification of his arteries. I think some little depressing influence brought to bear upon the central organ was the final cause of the outbreak of gangrene; it was the last straw which determined the occurrence of the condition. But I think it was a case of acrosphacelus chiefly from disease of the arteries; but its occurrence symmetrically in both hands simultaneously leaves us to seek for something a little more than that, although he had not noticed it, which was brought to bear upon his general health, which produced the outbreak.

Lastly, I have to mention another curious form, which I daresay some of you may be familiar with, but if not I would again say that you should make a note of the diseases which you wish to see, and not be happy until you see illustrative diseases which find their names in books, and see for yourselves examples of each; so I would say ask in the department of skin diseases to be shown a good example of the diffuse scleriasis of the skin or diffuse morphœa. I will try to describe it to you as well as I can in words.

It consists in this: that the hand, instead of remaining plump and red, as it should be, with the skin flexible, becomes stiff and hard, the fingers are stiffened and so is the subcutaneous cellular tissue, the ends of the fingers become less plump, they look empty of blood, and the hands look like badly-coloured pieces of tallow more than anything else, with patches of blood here and there, and in advanced conditions the hands are useless owing to this scleriasis. It is an indurated condition of the skin and subcutaneous cellular tissue. This will extend up the arm to a certain distance, the distance varying according to the severity of the cases, passing off very gradually, and recognisable for a considerable distance in the majority of cases. Diffuse morphœa was the old term which we used

Digitized by Google

to discriminate between this condition which is not distributed by the nerves, from another form of morphœa in which the skin passes into this hard condition in which it goes in streaks, just like herpes zoster or like shingles, and is clearly distributed by the agency of the nervous system. In these cases all the digits are alike, but the digits of the toes are less affected than those of the upper extremity, and the face is affected. If you look at a patient who has this sclerodermia as it is called, if you look in the face of a woman who has this condition—and it nearly always occurs in women—you notice that her mouth is a little open, because the lips have become hard and rigid. The skin of her face looks glossy and a little too smooth, and it is stippled over with little punctate vessels, or perhaps like little bits of seaweed scattered over the face. If you were to take hold of the skin of the face of such a patient you would find you could not pinch it up, because it has become all hard and stiff. This absolute inability to pinch either the face or the fingers is a characteristic of this condition.

You will have gathered from what I have said that the condition is a very definite one and very characteristic indeed; and if you have seen and investigated one case, you will not fail to recognise another. That is a condition of sclerodermia or diffuse scleriasis of the skin, or as some term it, a diffuse morphœa. With those cases you almost always get the history that the patient is liable to more or less paroxysmal change, that sometimes the hands are worse than at other times, but that they are never well, and indeed the condition never gets anything like well. Then acrosphacelus occurs, the bulbs of the fingers are all contracted and hardened, and little bits of gangrene occur at the ends of the fingers close under the nails. That is a condition constantly observed. Sometimes the whole end of a digit will pass into gangrene. This condition is allied to Raynaud's phenomena, but it is one of those which justifies me in saying what I did, that we must not speak of Raynaud's disease, but of Raynaud's phenomena: because these cases, although Raynaud's phenomena occur in them as part of their manifestation, they are quite distinct from the rest. They stand at a very considerable distance from the cases in which the patient can get well. These cases practically never get well; they go from bad to worse, and the only modification of that statement must be that in ten or fifteen years sometimes, a little improvement may take place as the result of treatment. But they never get absolutely well. So these justify me in saying that there are different groups of cases which should not be roughly thrown together under the name of Raynaud's disease. Raynaud himself described some of these cases of sclerodermia in his first thesis, and I think it is rather a pity that these sclerodermia cases get confused with the others, because they constitute a group apart. Still we must not simplify too much, we must not be too arbitrary, and make classifications in the same way that we would classify plants and animals according to species; that is not to be done with pathology, for the phenomena run into each other, and causes may interlace and make partnerships. And so we find relationships which we must recognise, and not differentiate too strongly and too definitely. So I assert, respecting this form, that you will almost always find that paroxysmal phenomena do attend it, and that it is exceedingly probable that the whole of the changes induced in the skin in a case of sclerodermia—induration and a hard state of the skin—are induced through the nervous system, and in that way they have to do with the paroxysmal forms of Raynaud's phenomena. But those paroxysms are sometimes of greater power; sometimes there is rigidity, sometimes there is a tendency to gangrene, and sometimes there is a fair state of warmth and comfort; they vary in the individual. And in that way they correspond with other forms of the disease under consideration.

I think I have now asserted several points. I have described the juvenile form as one which is definitely paroxysmal, and in which extreme lividity and blueness of the extremities is noted, and is one of the chief conditions. Then I have alluded to the senile form, in which pallor is more usually the condition. I may say a word or two more about the senile form of Raynaud's malady. In the senile form—and I think I have already suggested that—we expect structural organic diseases, and very possibly diseases of the heart and especially of the arteries. I have two or three very instructive cases of this senile form. One, a gentleman very well known in London, who has very extensive engagements, who has hitherto

Digitized by Google

enjoyed good health but never a perfect circulation, who drives himself amongst the streets of London, and therefore must have a fair circulation in the hand to do that, has always suffered a little more than usual from cold hands when holding the reins. He has reached the age of sixty-two; he has lived freely, he has had his fair share of champagne and meat, but he has had every advantage as regards health, except, perhaps, that he has had some over-work, and I think, probably, he has taken stimulants rather more than was good for him. Now you can imagine the condition of the fingers of a corpse which has been dead twenty-four hours, and when he held up his fingers I, when he was not noticing me, took careful note of their appearance, and they looked painfully like those of a corpse; the bulbs of the fingers were dry and shrivelled, so that you could not press them, and with little blue marks round the ends of his nails. It was evident that his fingers were on the verge of going into gangrene. His pulse was feeble, but not remarkably so. Whether there was calcification of his arteries was a little doubtful. I thought there was calcification, but it was certainly not present in extreme degree. I refer to this case so as to mention a new symptom which I have recently observed in reference to these phenomena. He told me—and you must always listen to what a patient tells you, and carefully examine what he says, because your patients will often show you things which you might otherwise overlook,—he said, "I have got a red mark in my nails." I found that what he meant was a little line exactly where the nail leaves its bed, a line of redness very conspicuous indeed, which was present in all his nails just where it was about to leave its bed. This redness could not be removed by pressure. It was quite clear that he had got a condition of thrombosis of the line of the capillaries at the very ends ot his nails. I am interested in collecting portraits of nails, and I took up two other portraits, which have been made for me of other cases of Raynaud's phenomena, and I found my artist had accurately represented this little red fringe which I had overlooked, but there it was in the other portraits. I think we may add that to our symptoms of Raynaud's phenomena.

I ought not to detain you longer with this individual case; it is a good instance occurring in a gentleman who has had every advantage of life, who has not been unduly exposed to cold, and in whom gradually this happened, and his fingers have all but gone to gangrene. He has been under treatment for several months, and we have put him to bed, and I am afraid he will lose the ends of his fingers. Our object in putting him to bed was to keep him at an equable temperature and to favour the action of his heart and the distribution of his blood; so I hope he will escape actual gangrene.

Now two or three words as regards the general treatment, and then I have done. The preliminary to all speculations as to treatment is a thorough understanding of the causes of diseases, and it is no use, for the most part, going in an empirical manner—though we may develop empirical laws which the person may act on without any comprehension or any due understanding of the illness; but if we wish to advance therapeutics we must take into consideration all details of causation of disease. That is an obvious thing which I need not insist upon. The application of the subject amounts to this:—I have tried to establish the fact that there are different causes for Raynaud's phenomena, so all those must be taken into consideration in developing the treatment of it. If it be that the nervous system is very susceptible in a reflex manner to the influence of cold, the chief method of treatment will be to protect the individual from the influence of cold. And in young children, those who have the juvenile form, the main measure of treatment is to insist upon warm clothing, and if possible to insist that they must live in a warm climate. I have asked you also to study diseases of the lower animals and of plants; we want information very much as to international medicine. Many of you go abroad in the vacation; let us all keep our eyes open and learn what we can with regard to the influence of climate. It would help us very much now if we could tell whether Raynaud's phenomena are very much less frequent in the tropics than at other places. We know symmetrical gangrene occurs among the Hindoos occasionally, but whether a patient liable to this paroxysmal form, especially a young patient, in whom the nervous system would be susceptible to the influence of external cold, would be benefited as a rule by going to the tropics is what we want to know. My suspicion would be that they are, and I always in private practice try to send a patient

Digitized by Google

who is liable to Raynaud's phenomena to winter in Egypt; not to a tropical climate but to a warm climate, and I can speak in clear terms as to the benefits that patients have derived from habitually spending the cold part of the year out of England. If that cannot be done, there should be every precaution in the shape of warmth, warm clothing to any extent, to whatever extent it is necessary, to protect them from the injurious influence of external temperature. If it be one of scleriasis or morphœa, the cause is much less easy to assign, but here again the paroxysmal nature of his illness implies the susceptibility to external influences of cold to some degree. If the patient be an adult who cannot go away during the cold part of the year—and this remark applies to all forms of gangrene in the extremity—the very best remedy for preventing undue susceptibility in regard to reflex circulation and maintaining the circulation in the extremities is opium; and the greatest good I have been able to do to patients who have remained in England has been by insisting upon the constant use of small quantities of opium. If purgatives be given, then small quantities of opium do not agree; I mean five drops of laudanum, or three or four drops as a sedative given three or four times a day, with tonics and careful attention to the liver. I have seen young patients improve very much and remain exempt from disturbance of the circulation afterwards. Those are the remedies which I speak of with the greatest confidence in all morphœa patients. Let them take a minute dose of opium, and they will be the better for it. Other remedies must be adapted to the needs of each case, tonics if necessary. There should be also I think a moderate use of stimulants,—and that is a very important measure; but they must be given with very great care, because the use of alcoholic stimulants is liable to be followed by subsequent contraction of vessels leading to a much greater resort to it after the use of opium.

In support of this beneficial effect of opium, some of you who may have watched an accidental case of poisoning by opium may have noticed that if the patient is dying from an overdose of opium, he may be pulseless almost and yet may maintain his temperature; his hands may remain warm, or even hot, at the time of his death.

Raynaud himself insisted, in the latter part of his life, a great deal upon electricity. I have tried galvanism according to his methods of recommendation in competition with opium; and I would say again that my faith is in the use of tonics, the maintenance of the external temperature, and the exhibition of small doses of opium for a long time; it may be even extended to years.

SPINAL symptoms produced by tea intoxication are not reported, at least in the literature for the last five years. All reported cases have reference to cerebral symptoms. Wood ('Medical News,' 1895) reports a case where theism produced headaches, neuralgia of face and neck, attacks of vertigo, mental disquietude and confusion, forgetfulness, periods of despondency, restless sleep, nightmares, dreams, hallucinations. Cooney ('Indian Medical Record,' Calcutta, 1897) observed palpitation of the heart, headache, giddiness, mental depression, melancholia, suicidal monomonia. Castle ('Cincinnati Lancet-Clinic,' 1886) experimented on himself with theine, and stated that besides local loss of sensation he had psychic disturbances, confusion of ideas, excitability, fainting spells. Thomas J. Mays ('Theine in Treatment of Neuralgia,' Philadelphia, 1888) has not observed symptoms of cerebral intoxication, but he did observe symptoms of anæsthesia. Lehman and Tendlan ('Arch. f. Hyg.,' München, 1898) undertook a long series of experiments, and stated that 20 to 40 grammes of tea produced muscular spasms, muscular fatigue, muscular restlessness and tremor, giddiness. Amblyopia was noticed in tea intoxication by Campbell ('Lancet,' 1898). We therefore see from these few cases that there has been no mention of spinal symptoms. Shall we reject the possibility of influence of the elements of tea on the cord?

Theine and essential oils of tea are poisons to the nervous system. On the other hand, Schwann demonstrated that tannic acid will leave a precipitate from digestive elements, and render them inert; atony of bowels and stomach is the result; auto-intoxication might follow. Cooney says that black lead is used to give a bloom to black tea. We therefore see that three sorts of poisons, if not more, take part in intoxication by tea. If other intoxications, like alcohol, lead, mercury, etc., are apt to produce spinal symptoms with involvement of the cord, we believe that the same might be expected from tea; it seems to me there is only a difference in degree.—Dr. ALFRED GORDON, *Therapeutic Gazette*, July 15th, 1901.

Digitized by Google

WITH MR. STANSFIELD COLLIER AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

Some Cases of Congenital Talipes.

Gentlemen,—This four months old baby is, as you see, the subject of talipes equino-varus of the right foot. The foot is inverted and adducted; its inner border is concave and appears short, its outer border is convex, and is relatively long. The concavity of the inner border is prolonged into the sole, to end in a deep crease beneath the posterior tarsal joints, and the external convexity is continuous with the exaggerated dorsal curve. As I endeavour to reduce the inversion you see more plainly that there is over-extension at the ankle-joint. It is clear that the deformity is affecting chiefly the tarsal joints and bones and the ankle-joint, but notice that there is also marked deviation at the metatarso-tarsal and metatarso-phalangeal joints.

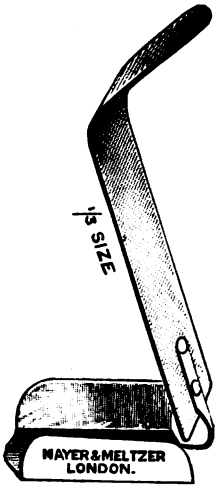


Fig. 1.

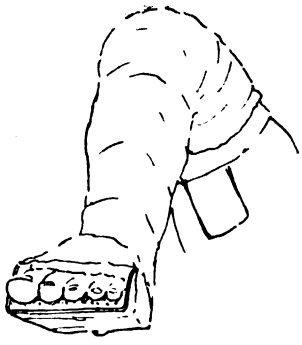


Fig. 2.

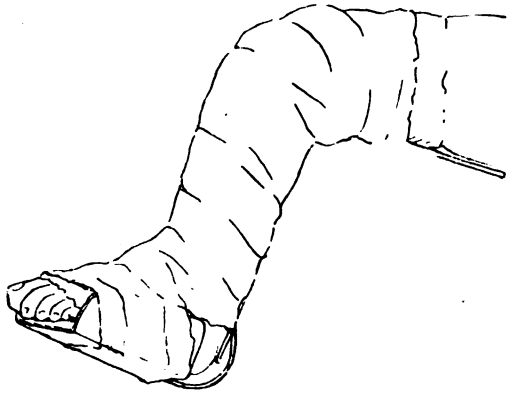


Fig. 3.

Compare the talipedic foot with its fellow. The affected foot is smaller, and seems to be especially wanting in length. The shortness will be much less marked, of course, when the foot is unfolded in the process of cure. You see at once how ill-developed the calf muscles are, and there is another thing I particularly want you to notice, which is very easily appreciated in this case, because the child is very thin; it is that the inversion is not limited to the foot, but affects the other parts of the limb. This is an excellent example of that common accompaniment of congenital talipes equino-varus, which is called crus varum.

As I bend up the limb we see how closely it fits against the child's trunk, and can well believe that it was in this cramped posture that the fœtal limb grew. We may compare this with the static deformities produced in post-natal life, and look upon the club foot as the anatomical expression of long-continued maintenance of this position; nor need we think that the rudiments of the limb were inherently destined to grow in this deformed fashion.

One can well imagine that a fœtus, untrammelled, and endowed in a due degree with *savoir vivre*, upon finding its limb squeezed between its body and the wall of the amniotic cistern would leap into a more comfortable position.

This child may have been embarrassed by want of space in the womb, or fettered by amniotic adhesions or a twisted cord, though there is no evidence of abnormal environment. Now and then—too often to be explained by coincidence—one finds the deformity present in father and child, and you may make of this an argument against the view that the position of the limb *in utero* is all-important in the production of the deformity. If you do so I shall take refuge in the possibility that the essential inheritance is a lateness in perfecting the important fœtal property of irritability.

To return to the examination of the foot, although

Digitized by Google

the deformity is considerable this is not a severe case, because upon manipulation you see that the short structures yield considerably, and if you call to mind how little advanced the ossification of the tarsus is at this age, you will allow that the bone rudiments are capable of being moulded to some extent.

The physical properties of pliability and of plasticity will make it easy to improve immediately the outward shape of the foot, and the influence of altered circumstances upon the processes of growth will gradually effect a cure.

The splint which I prefer for these cases is made of thin zinc-plated iron, not too flexible. The piece to be applied to the sole is rather longer and wider than the foot, and its lateral edges are turned up. The shaft of the splint, of the same width as the sole piece, is to be applied to the back of the limb, and to extend to the middle of the thigh. The two parts of the splint are connected at the heel by a stout copper wire, pliable enough to allow of the footpiece being brought into any required relation with the shaft. At the level of the knee one bends the splint to an angle of about 110°.

After manipulating the foot so as to stretch the resisting structures, the limb is wrapped in a flannel bandage which has been sprinkled with boracic powder. The footpiece is now bent so as to fit the sole in the deformed position, and the splint is applied. The backpiece and thigh being grasped with the left hand, the footpiece and foot are steadily brought into an improved position. A strip of poro-plastic felt of about two and a half inches in width, long enough to reach from the toes to the middle of the thigh, is softened by heat, and applied to the front of the limb. I now fix the apparatus with an ordinary bandage and a cross-gartering of strapping.

The child should be brought here twice a week for manipulation and further correction, and the aim will be to get the foot into the position of calcaneo-valgus as soon as possible, and to keep it so until the child is of sufficient age to walk, by which time we may expect that the deformity will be cured. If the mother were sufficiently teachable, I should allow her to remove the apparatus, and to manipulate the foot daily.

If the short structures were less extensible, I would have the baby anæsthetised, and divide subcutaneously the tendo Achillis and the posterior fasciculus of the external lateral ligament, and, if necessary, the resisting structures of the inner part of the sole. Several stretchings under anæsthesia might be required. In this degree of severity I employ the metal splint, with a plaster-of-Paris bandage. Now that the splint is applied you see that the limb falls into the position of external rotation, and this seems to me to have the advantage of tending to correct the crus varum. This splint is satisfactory in out-patient practice, because one can hardly produce swelling of the toes or pressure sores with it, and because it is inexpensive.

It would be unfair if I did not show you some unsuccessful cases, of which I have my full share. If you cross-examine the mothers of some sorry examples which are waiting in the next room, you will, I think, find in each case that the real cause of failure is foreign to the victim, and that there has been some obvious defect in the management of the deformity. As a rule the poor mothers are not to be blamed, for circumstances make it impossible for them to attend regularly, and very rarely are they wanting in solicitude. We must be very earnest in cautioning them against delay and interruption of treatment, and must explain to them that the process of improvement is necessarily gradual, and that an operation is only the beginning of a cure.

Now will you look at this child of two and a half years. It was brought to me when it was ten months old, and remained under treatment for eleven weeks, and the mother ceased to bring it because it had whooping-cough. The deformity is very marked, and there is some bursal thickening over the outer part of the cuboid and os calcis. The tarsal joints are rigid, and the outer part of the foot feels massive as compared with the inner part. What treatment shall be adopted? We might subcutaneously, or by the open method, divide the soft parts of the inner side of the foot, and thus give the inner border of the foot its proper exterior shape and proportion, and to do this we should have to make a wide gap between the scaphoid and the astragalus. But would the gap be permanent? I think not. Then will this measure influence the growing of the bones so that the correction will endure? This seems too much to hope for. A cuneiform osteotomy, with division of the tense structures, may be relied upon to give

Digitized by Google

a good result, provided that the after-treatment be not neglected. Excision of the astragalus would be unsuitable, because the deformity is unilateral.

As you examine the feet of the three boys who are sitting upon the couch, the results of treatment appear satisfactory. But as they walk you see that only the first of them has a good gait. The second limps because the talipedic limb is shorter than the other, and with a thick sole you see that he walks pretty well. The third boy's gait is very disappointing, and is to be explained by persistence of crus varum. I shall order for him some walking instruments such as are figured in Mr. Jackson Clarke's work.

A Case of Circumcision.

This baby was circumcised by Mr. H. J. Curtis four days ago, on account of difficult micturition due to phimosis. The boy has done well, for the mother tells us that he has not seemed to be in pain and has slept well, and looking at the part you see that there is scarcely any swelling or redness.

Let me give you the details of the method employed. The skin of the prepuce was drawn forward between the handles of a pair of sinus forceps. The forceps were held obliquely, so as to avoid cutting the frenum. The prepuce was grasped by the forceps' handles at a chosen level, and section was made in the usual way with a scalpel. The parts removed were the outer layer of the skin up to the orificial ring, and a small portion of the middle layer, containing the larger veins which drain the prepuce.

The reflected layer of the prepuce was now slit along the dorsal middle line from the orifice nearly to the sulcus, and the two halves of it were turned down. With squint scissors the flaps were cut away at about a twelfth of an inch from the sulcus, except inferiorly, where a small tab of the reflected layer receiving the frænum was left intact. In the second stage, then, the patient was deprived of most of the reflected layer of the prepuce and a second portion of the middle layer. The upper part of the middle layer of the prepuce still remained. Consisting of delicate areolar tissue, and having no great bulk, it was inconspicuous until the retracted sheath of the penis was drawn forward, when the oozing tissue formed a small collar. This was seized with forceps and removed with scissors, care being taken to leave no loose tissue attached to the frenal tab.

The bleeding points were vigorously pinched with Well's forceps, and the artery of the frenum and a branch of the dorsum were tied with catgut. The edges were brought together by interrupted sutures of the finest catgut, as little tissue as possible being included in the stitches. The part was now smeared over with egg albumin, holding in suspension boracic acid in the proportion of thirty grains to the white of one egg.

The mother was ordered to re-apply this twice a day, and to apply no other dressing. She was also told not to pin up the napkin, and she was directed to make a pasteboard shield.

I would like to lay stress upon the importance of removing thoroughly the middle layer of the prepuce, and thus avoiding mechanical œdema, which must add something to the pain of the child and much to the distress of the mother.

For the sake of the patient's present and future comfort, I may warn the beginner against a too extensive removal of skin, and against sacrificing the frænum.

Medical Treatment during the Adolescent Period.—Edward Rosenthal writes that the adolescent period in the female may be said to be as critical in result as the menopause. The commonest symptom that presents itself is the one that refers itself to the menstruation. The cases may be divided into those who have never menstruated and those who have had a slight menstrual flow at infrequent intervals—once in six or nine months. The other symptoms are digestive disorders, headaches, languor, flushing, sensations of fulness in the abdomen, disturbed or unnatural sleep, or sleepy condition during the daytime, and some cutaneous affection—most commonly acne. All or part of these symptoms may present themselves. In cases where menstruation has never appeared an examination should first be made to ascertain if any anatomical reason exists, such as an impervious hymen or cervix, or absence of uterus, ovaries, or vagina. In such cases surgical measures are the only resources. Where no anatomical reason exists, much can be done to aid a cure. The absence of anæmia need not preclude the use of iron.—*Amer. Journ. of Obstetrics*, August, 1901.

Digitized by Google

TWO CASES OF SWELLING OF THE EYELID OCCURRING AFTER A BATH.

By THEODORE FISHER, M.D., M.R.C.P.,
Physician to Out-patients, Bristol Hospital for Sick Children, and Pathologist to the Bristol Royal Infirmary.

Soon after I was qualified, but while still a medical student, one of the servants in my father's house awoke one morning with well-marked swelling of the eyelids. I was consulted as to its meaning, and as to whether medical advice were necessary. We had subscribers' "notes" for a dispensary with which a small hospital is connected, and having in view in my mind the possibility of some kidney affection, I recommended that the girl should be sent to the dispensary for examination and treatment. She returned saying that the doctor had told her that the swelling of the eyelids was due to coughing. I was somewhat sceptical about this diagnosis, but whatever the cause may have been, it obviously was not Bright's disease, even in its mildest form, since at the end of two days all swelling had disappeared and the girl was in her usual health.

This was my first meeting with one of those minor ailments or symptoms of no serious import, which writers of text-books, or even teachers of medicine, either forget or consider not worthy of mention. Slight degrees of swelling of the eyelids, it is scarcely necessary to remark, are not by any means uncommon in some people when a little out of health. Such a peculiarity may run in a family. A short time ago a boy was under my care in the out-patient department of the Bristol Children's Hospital, who had suffered from occasional swelling of the eyelids. The boy's mother stated his father and his father's sister had throughout their lives been affected in the same way. When the swelling occurred in the father's eyelids he would complain of being unwell, to use his wife's words, he would describe himself as feeling "all gone." Our sense of general well-being is largely dependent upon the efficient working of our vaso-motor system, and the association of a feeling of tiredness with swelling of the eyelids suggests that the swelling is in some way dependent upon vaso-motor disturbances. Recently I have had two cases under my care which seem to support such a view. In two children swelling of the eyelids has followed a bath. In one it several times was brought on by a warm bath, and in the other occurred after a swim in cold water.

The first case was in a girl æt. 9, who was taken to the Bristol Children's Hospital on February 4th of this year for an attack of simple jaundice. It was mentioned that several times during the winter the child's eyelids had swollen after her Saturday night's warm bath. One Saturday evening, a week or two later, I called at the home of the child in the hope of seeing the swelling. Unfortunately, on that occasion there was no trace of it, but the remarks of a neighbour who was called in to support the statements of the mother were of some interest. This neighbour, an intelligent elderly woman, said her mother had died of disease of the kidneys, and when she first saw the girl with her eyes swollen, she exclaimed, "Why, she's got the dropsy," and immediately proceeded to examine the child's legs, by pressing over the shins in the way she had learnt from her mother's medical attendant. Her idea of the dropsy proved, however, to be unfounded. An observation of the baker's boy was also quoted as evidence that the swelling of the eyelids was not imaginary. He had seen the child one Saturday afternoon with her eyelids swollen, when she had been given her bath on the previous Friday evening. On the following Saturday he noticed the difference in her appearance, and commented upon it by remarking, "What a pair of eyes she had last week." Shortly after this visit I was able to see for myself that the swelling did really occur and was considerable in degree. I had asked the parents to send me a telegram when the eyelids again became affected. One Saturday evening the telegram arrived, and an hour later I saw the child. The upper and lower eyelids were then much swollen, especially on the right side, where the lower eyelid equalled half a large walnut in size and much resembled it in shape. Some photophobia and slight lachrymation were present. The swelling had commenced about half an hour after the bath, and I learnt on inquiry later that it lasted until the middle of the following day.

Why the eyelids should only swell occasionally, after a bath in this case, is not obvious. The mother asserts most positively that any marked variation in the temperature of the water has not

Digitized by Google

been the cause, and the child, who takes an intelligent interest in her own condition, supports her in this view. No variation in the time of taking food or in its character which is worthy of notice appears to have been present, neither has there been any indication of dyspeptic disturbance. Last April a few notes of this case were read before the Society for the Study of Disease in Children, when Dr. George Carpenter suggested vaso-motor disturbances preceding the onset of menstruation might underlie the condition, and Dr. Sutherland asked if there was any history of asthma in the family. Inquiry upon the second point fails to show that any spasmodic affection, such as asthma, or nervous complaint like epilepsy, has been present in the family. With regard, however, to the influence of changes in the organisation preceding menstruation, one cannot but think that they probably play some part in predisposing to the swelling of the eyelids. Yet the swelling does not occur at regular intervals. Sometimes only a week may elapse before the swelling returns, or the child may be unaffected by her bath for five or six weeks. It seems possible that sudden changes in barometric pressure may predispose to the swelling. There has generally been marked alteration in the character of the weather about the time the swelling of the eyelids has occurred. The weather has sometimes been mild, sometimes cold, but the change from one to the other has generally taken place very shortly before the onset of the swelling. It should be mentioned, however, that the time of year does not seem to exert any material influence. The eyelids were first affected last October, yet the attacks have continued through the spring, and at the time of writing I find that one occurred recently, in the third week in June. All one can definitely say is that whatever the predisposing cause or causes may have been, the exciting cause has invariably been a warm bath. One negative fact in connection with the case should be mentioned. In several cases of occasional swelling of the eyelids in children I have found cyclical albuminuria to be present. The urine of the child, however, taken from different times of the day, both during occurrences of the swelling of the eyelids and on other occasions, has been examined without the discovery of the faintest trace of albumin.

In the other case which has come under my notice, the exciting cause of the swelling of the eyelids has been a cold bath, and marked intermittent albuminuria has been present. This case was also in a girl—but rather older—aged thirteen years, who attended the Bristol Children's Hospital on June 8th, 1901, for swelling of the eyelids. Two days previously she had had a swimming bath, the first taken this year, and on waking the following morning the eyelids were much swollen. The preceding summer she had frequently visited the swimming baths, but the bath had never been followed by swelling of the eyelids. The swelling was still present in considerable degree when she was first seen, but had nearly disappeared two days later. After the swelling had disappeared slight yellowish discoloration remained. The girl was pale, but did not complain of feeling unwell. There had been slight headache, but her appetite was good, and there were no symptoms pointing to disorder of the digestive organs. Two days later three specimens of urine taken from different times of the day which were brought were examined. There was no trace of albumin in the morning urine, but it was present in considerable degree in both the mid-day and evening specimens. While not even a faint ring was obtained in the morning specimen on testing with cold nitric acid, the other two specimens not only gave well-marked rings with cold nitric acid, but after boiling, and being allowed to stand for three hours, the deposit was about a fifth in one, and a sixth in the other. Tested with Esbach's albuminometer, the evening specimen gave ·1 per cent. of albumin. The mid-day specimen was not tested quantitatively on this occasion, but two days later another specimen gave ·7 per cent. of albumin. The quantity in the evening urine had then sunk to ·075. Examination for casts was twice made, but none were found. An examination made three weeks after the onset of the swelling of the eyelids showed that the amount of albumin in the mid-day specimen had sunk to 0·25 per cent., and that the quantity in the evening urine had become too small to be estimated by Esbach's albuminometer. A fortnight later, that is five weeks after the onset of the swelling of the eyelids, the mid-day and evening urines still contained some albumin. Since albuminuria has been present in this case, I have thought it wise that the girl should discontinue her swimming baths.

Digitized by Google

We shall not therefore have an opportunity of seeing what would be the result of another cold bath. Whether the intermittent albuminuria in this case was entirely due to the cold bath, or merely aggravated by it, is difficult to decide without prolonged examination. Since the amount of albumin in the mid-day urine sank from ·7 per cent. to ·025 per cent. in less than three weeks, it is obvious that the bath at least increased the albuminuria. The fact, however, that in other cases of swelling of the eyelids I have found the intermittent albuminuria persisting as long as one and two years after the first examination, leads me to think that in this case also it is probable that albumin is always present in the urine at some time of the day.

One point connected with swelling of the eyelids is of some interest—that is, the presence of discoloration after the swelling has disappeared. It has usually been present in some degree in the first case, but was better marked in the second. In some cases it may be so distinct as to resemble a bruise. The mother of one child told me that the appearance of bruising which followed swelling of the eyelids frequently led to the suggestion by friends that the child had received a blow. That marked discoloration of the eyelids may follow swelling of the eyelids of vaso-motor origin is suggested by a case in which swelling and subsequent discoloration of the eyelids was associated with epileptic fits. A woman who was bringing her baby to the Bristol Children's Hospital had one day her lower eyelids of such dark colour, that my notes describe her in appearance as closely resembling the Kabyle women of North Africa, who blacken the lower eyelids as a part of general adornment on festive occasions.* On inquiry I found that the woman suffered from epileptic fits, and that after a fit her eyelids were said to swell. When the swelling disappeared discoloration remained.

Occasionally swelling of the eyelids in children will be found to be associated with an erythematous rash on some other part of the body, and in these cases, doubtless, the swelling is of erythematous nature. Such swelling of the eyelids generally lasts for two or three days or longer, and after disappearing does not recur. Erythematous swelling of the eyelids may affect one side only. Other instances of swelling of the eyelids on one side may sometimes be of puzzling character. For example, in a child aged three years swelling of the left eyelids associated with headache appeared to have been due to inflammation of the left tympanum, since as soon as discharge from the left ear appeared the swelling of the eyelids subsided. It is scarcely necessary to remark that swelling of the eyelids on one side may be a symptom of thrombosis of the cavernous sinus. Such a condition is rare and generally fatal. I have made an autopsy upon such a case. The thrombosis was of septic character, but the source of infection could not be found.

In conclusion, it may be remarked that many cases of swelling of the eyelids seem to be largely dependent upon what may be somewhat indefinitely termed personal physiological peculiarities—that is to say, while one person when not feeling well may present a sunken appearance of the eyes, in another the eyelids may show some degree of swelling. It is interesting to note that in some of the cases in which swelling of the eyelids occur, cyclical albuminuria may be found. This form of albuminuria was present in one of the above cases. The special interest of these two cases lies perhaps in the fact that a definite exciting cause for the swelling was present. Since in one case, however, cold baths had been taken before without the swelling of the eyelids having occurred, and in the other case not every bath occasioned the swelling, it is obvious that personal peculiarity and an exciting cause need not be sufficient to lead to this condition. The presence of at least a third factor may be necessary, but the nature of this third factor in the above cases was not clear. Looked at from the medical standpoint, swelling of the eyelids may be of simple nature, yet physiologically the reason for its appearance may be obscure.

* This is not peculiar to Kabyle women; it is, I believe, an ancient and common custom amongst eastern women. In North Africa, however, the Kabyle women, although Mohammedans, do not wear veils, and consequently men may have the good fortune, as has fallen to my lot, to see them decorated for some ceremony.

Dermoid Cysts in a Child.—Legueu, operating on a child for supposed appendicitis, found instead dermoid cysts of both ovaries, the pedicle of the right having undergone torsion, causing the symptoms.—*Soc. de Chir. de Paris*, April 24th.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 463. WEDNESDAY, SEPTEMBER 11, 1901. Vol. XVIII. No. 21.

CONTENTS.

PAGE

*A Clinical Lecture on the Surgical Treatment of Tuberculous Glandular Affections of the Neck. By H. T. BUTLIN, F.R.C.S., D.C.L. 321

*A Clinical Lecture on a Case of Muscular Atrophy. By J. H. BRYANT, M.D., F.R.C.P. 327

*Clinical Remarks on Four Cases of Intra-ocular Hæmorrhage. By W. M. BEAUMONT, M.R.C.S.... 335

*Specially reported for the Clinical Journal. Revised by the Author.

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

THE SURGICAL TREATMENT OF TUBERCULOUS GLANDULAR AFFECTIONS IN THE NECK.

Delivered at St. Bartholomew's Hospital, June 5th, 1901.

By H. T. BUTLIN, F.R.C.S., D.C.L.

GENTLEMEN,—To-day the subject of my lecture is chosen for me. Two or three weeks ago the editor of a well-known medical paper told me he would like to publish the clinical lectures which I delivered this Session. It occurred to me that he might perhaps be useful to me, and I said, can you suggest to me a number of subjects which would be suitable for me to lecture on, and which at the same time would be useful to the readers of your JOURNAL. He thereupon sent me a list of subjects, and, curiously enough, three out of the eight or nine subjects he put down I have lectured on in the course of the last two or three years, and I think they have been published in the 'St. Bartholomew's Hospital Journal.' But amongst the subjects which he set down on his list was the surgical treatment of tuberculous glandular affections, and it struck me that that was a very good subject, as tuberculous glandular affections are constantly treated here in the hospital and in private practice. It is therefore of interest both to students and to men who are in practice. On looking at the list again, I saw that four words had been prefixed to the title—"'On some Points in' the Surgical Treatment of Tuberculous Glandular Affections." Evidently the editor thought the whole subject would be much more than could be got into one issue of his paper, and that it might take up two or three; accordingly he put some limitation on the subject. When I came to consider what those points should be, the first thing I determined was to limit the subject still further, and to speak only of the surgical treatment of tuberculous glandular affections *in the neck*, because tuberculous glands of the neck are very much more common than in other

Digitized by Google

parts of the body; they are often scattered widely over both sides of the neck; they are apt to undergo various changes which are very distressing to the patient, and the treatment of them presents peculiar difficulties, and sometimes more or less considerable danger. Therefore my subject to-day will be "On the Surgical Treatment of Tuberculous Glandular Affections in the Neck."

In order to have an idea of how the subject should be dealt with, I thought first how tuberculous glandular affections are recognised. Without going into the question of diagnosis to any great extent, I may say that although tuberculous affections of the glands are to be found in persons of every age, from infants to old people, yet they are very much more common in young people and in children. I think I may go so far as to say that of chronic affections of the glands in the neck which occur in children and young people at least nine-tenths are tuberculous. Where the glands suppurate or caseate, and the suppurations are very slow, where ulcers form which have typical characters, where sinuses are left in the neck, all these things speak clearly of the tuberculous nature of the disease. And again, the patient may present other signs of tubercle. The affection of the glands may be associated with enlarged tonsils or adenoids, or with both, or with some other trouble about the throat, which is of the low tuberculous type.

The next point is, what course tuberculous glands run, and what results they may produce. Tuberculous glands may get well, and that is a very important thing to bear in mind; they may get well like any affection which is not tuberculous, and they may get well like tuberculous affections of other parts of the body, such as tuberculous disease of the bladder, of the throat, or even of the lungs.

In the second place, when the glands have become enlarged owing to the existence of tubercle in them, they may remain permanently enlarged for three or four years, or even longer, neither getting larger nor smaller in that time, nor yet indicating any of those various changes to which they are liable.

In the third place they may be inflamed, and you will hear not uncommonly that a gland in the neck which was small a few days ago has suddenly become larger and more painful and tender to the touch, and that the skin over it has become red. You may learn later that it has subsided and the inflammation has passed away. In the course of a few weeks or months the same thing may happen again. Further, tuberculous glands may suppurate, that is to say the inflammation may go on to suppuration, and it very frequently does so. The suppuration may be either in one place, as in this little gland which I send round, or it may occur in several places in the same gland. The glands may caseate, and they sometimes become calcareous: but I do not remember ever to have removed calcareous glands.

Instead of being limited to the interior of the gland, this process of inflammation and suppuration may spread to the surrounding parts, and then you have a peri-adenitis, that is, an inflammation of the tissues round the gland. In this the glands may become adherent to the surrounding parts, and may become adherent to very important parts, such as large vessels and nerves in the neck. The extension may so involve the muscles that it may be impossible to remove the glands completely without at the same time taking away a portion of muscle. The disease may make its way towards the surface, and the skin may be inflamed, and then you get a typical tuberculous ulcer of the neck, and sometimes a long sinus is left which leads down to caseous glands, or to a little cavity which continues to discharge for an immense time, perhaps for years.

Thus a great number of troubles are produced by tuberculous glands, and the patient may be more or less seriously disfigured, as you will see when you come to look at these patients who are waiting outside.

I see that my editor has put down "On the *Surgical* Treatment of Tuberculous Glandular Affections," by which I judge he thinks there is some other kind of treatment of them, or, at all events, he thinks that people may believe it possible to cure tuberculous glands by some other course than surgery. Nowadays everything is surgical, everybody wants an operation done, and wants to be cured right off. But as a matter of fact tuberculous glands are treated, usually for a long time, and sometimes for too long a time, by medical measures. They are treated by fresh air and careful dieting, and cod-liver oil and steel, and a variety of other things, and I believe that in the first instance almost every tuberculous gland and

Digitized by Google

swelling in the body is treated with some application of iodine.

Then comes the question as to when tuberculous glands should become the subject of surgical treatment, and after that what the surgical treatment should be. As far as I am personally concerned, and I suppose this applies also to most persons who are engaged in surgery and not in medicine, the answer to that question is very simple indeed; if a patient is brought here to me at the hospital, or to my house, with the diagnosis of tuberculous glands already made, the assumption is that such a patient is ready for surgical treatment. And that is the fact. By that time all kinds of medical means have been tried and have failed; everything which could reasonably be done has been done, and the time has arrived when surgical treatment begins.

I went over to myself the various conditions in which I have seen these patients for the first time, and from that, of course, I judge of the condition at which glands have arrived before they are regarded as fit subjects for surgery. A few months ago I was sent for to see a little girl who had a number of glands in the neck. For a long time she had had a mass there half as large as my fist. She had had various tubercular manifestations at different times. As she was the only child there was a great deal of trouble and anxiety about her, so every sort of medical means had been employed for many months. Then her temperature began to rise to 102° and 103°, and the child was very poorly indeed. Then the glands became more swollen and very tender, and though the skin was not in the least affected, a surgeon was called in, because it was felt that the time had arrived for surgical interference. I cut down and removed those glands, and found that although some of them were adherent they could not be got out. But they were full either of caseous material or of pus, and some of them were actually changed into bags of pus and pockets of pus, with nothing of the gland structure remaining.

I may mention a similar case which I was called to in London just before I went for my holiday. I operated upon a young lady and removed her glands. She had no sinus, or ulcer, and the skin was not affected. In the same house was a nursemaid, who had been in the family many years, and she had two or three enlarged glands in the neck, which had never been inflamed, which had been enlarged for a great number of years, and neither she nor anybody else could remember that they had ever changed very much. She watched with very great interest the result of the operation on the young mistress, and seeing that it turned out satisfactorily, she thought she would like hers done. She desired the operation in order to get rid of the disfigurement produced by the permanent enlargement of glands which were thought to be tuberculous.

Again, I saw several months ago a child who had one enlarged gland at the angle of the jaw, which every now and again inflamed and became larger and tender, and the skin became reddened over it. As this had happened several times the parents at last consented to have it removed by surgical means. That makes three conditions—*i. e.* glands which remain permanently enlarged; glands which rise and fall as a result of inflammation, but do not necessarily suppurate, although they may do so; thirdly, glands which have suppurated, but are not adherent to the skin, so that you can deal with them without cutting away any portion of the skin. Then, again, there are glands which have suppurated, and in which ulceration of the skin has taken place, or a sinus remains and does not get well. After injecting it and trying every palliative treatment which may result in cure, at length the surgeon is called in and an operation is performed, to get rid not merely of the glands upon which the trouble depended, but of the sinus and disfigurement of the skin. I have operated upon such a case in the last month. I think those are all the conditions of tuberculous glands in the neck which require surgical treatment.

Let me point out to you that tuberculous glands of the neck are almost invariably a secondary manifestation of the disease, not primary, and therefore you should always look, just as in any other kind of glandular disease, very closely at other parts to discover where the primary disease is. That primary disease should, if possible, be dealt with. For instance, enlarged tonsils and adenoids and ulceration on the scalp, and a number of other things, should be searched for, dealt with, and cured or removed if possible, so that the source of further infection may be got rid of; although even after removal of the primary source of trouble, the tuberculous glands may remain as they were before, or may even possibly grow worse.

Digitized by Google

I have often reflected to myself that we really want a Royal method of curing this disease. We want a Royal method by which we may get rid of tuberculous glands in the neck which require surgical treatment without leaving any scar, and in such a manner that there shall be no recurrence *in situ*, no affection of other glands on the same side of the neck, and no affection of glands on the other side. And if anybody will discover such a treatment as that, he will not only acquire a very great name, but he will make an enormous fortune. Up to the present time neither I nor anybody else knows any such method of treatment. Although the parents of children come and hope you will be able to tell them how this is to be done, you can only tell them, after all, that you will do your best to get rid of the glands which are obviously affected, and will do so with the least danger to the patient's life and with the least possible disfigurement, and that is the best you can promise. You can make no promise that other glands on the same side will not be affected, and you will certainly make no promise that those on the other side may not at some future time be involved in the same process, and that those groups of glands accordingly may not require the same treatment as the group which you are about to deal with.

Now I come to the object of this lecture—treatment. For the present it is reduced to a routine. But you must not imagine that the routine of to-day will be that of ten years hence, or that the routine of to-day is that which was in vogue ten or fifteen years ago, because it is very different. At the present time most surgeons adopt one particular method for the surgical treatment of tuberculous glands of the neck. That treatment is, in nearly all cases, excision. It sounds very simple indeed, and perhaps it is for the moment the most satisfactory surgical method. But I think it may be modified in certain cases, and I will take the conditions of glands I have described, and will tell you what I think is a suitable treatment in each of these conditions. Take a young person who has an enlarged gland somewhere here, just behind the sterno-mastoid muscle, very big. It is beginning to soften, and you make up your mind that it contains pus; you feel sure that it does. The skin over it is not reddened, the gland is tolerably deeply situated, and although it may be a little tender, still it is not seriously tender, or troublesome, except from the fear of what is going to happen to it. Instead of removing such a gland, as is the fashion at the present time, I have been in the habit under certain circumstances of attacking it by a very tiny puncture, and I wait usually until the gland has completely broken down, or at all events a good deal so, but I do not wait until the skin is involved or is reddened over it. I then tell the doctor and the friends that if they are willing to give up a very considerable amount of time to it, if they are not in a hurry to have it cured for perhaps two or three or more months, it is often possible to get rid of a single gland like that with practically no disfigurement. What I have done for years past has been to take a small knife, a tenotomy knife, and make a puncture into the gland which passes through the skin, subcutaneous tissue, and deep fascia. The puncture passes into the gland. Or I push a tolerably sharp-pointed director into the gland and squeeze out the pus along the director, and syringe it out with some antiseptic solution. Then I insert either a little strip of gutta-percha tissue or a piece of sterilised silk. Every day the cavity requires to be washed out, and the pus escapes; the pus should escape so readily that it does not require squeezing. This may have to be carried on for a considerable time before the disease gets well. You must point out that at any time this treatment may have to be given up and the gland may after all have to be excised, because the tuberculous trouble may extend along the opening to the skin; and if that is the case, the sooner the gland is taken out the better. Still, it is so successful that a good number of people have been treated in that way without leaving any perceptible scar. Of course, in people of what are called the better classes, that is to say the higher social grades, it is a matter of extreme importance that every effort should be made to avoid disfigurement, and if there must be disfigurement, it should be the very least which can be produced. I would point out that the best subjects for this kind of treatment are older children and young adults, because little children do not bear that day-after-day meddling with the part, and dressing and syringing which are necessary. The gutta-percha tissue or drain is liable to ooze out, and it may be difficult to re-insert it actually into the gland. If you are not going to treat the case throughout yourself, you need to be sure that the man who has

Digitized by Google

charge of the patient is not merely a competent man, but he should be a man like one of our house-surgeons here, who is determined to do what is necessary, and drain the cavity regularly and thoroughly, and to carry the thing through successfully, whatever trouble it may cause him.

The next class of cases is that in which you have a group of glands which are not inflamed, but which have been permanently enlarged for a very considerable period. The best thing possible is to remove them by such means as I shall speak of presently.

The third group of cases, those in which the skin is adherent and reddened over the tumour, may require, and in all probability will require, more than a mere incision of the skin; you have to cut away just that amount of skin which is inflamed, so that after removing all the glands you can bring the edges of the skin together in such a way as to secure a linear scar.

Last of all, there are those cases in which there is ulceration, or in which a sinus exists. In them you have to cut away all the affected skin, and you may have to undermine the surrounding skin to bring the parts neatly together after the removal of the glands, and you may have to sacrifice a good deal of the skin and to do a considerable operation to save the patient from severe disfigurement.

As to the method of removal of enlarged glands, my house surgeon, Mr. Rose, has been good enough to bring here the instruments which I am in the habit of using, and I will lay down for you certain rules which I believe, on the whole, are the best you can adopt in the excision of tuberculous glands, especially in young people. In the first place you should make your incision as far back, and generally as high up as possible. If, for instance, a quantity of glands are affected on the posterior border of the sterno-mastoid muscle, you can get them out through an incision quite at the upper part; and though it may be more convenient to make your incision in the lower part, you have to give careful consideration to the later result. A scar will be much less obvious under the hair than in the lower part of the neck, and that is of especial importance to a young lady, who does not want to be marked at the lower part of the neck. Next, you make at first a small incision, which you can always enlarge if necessary. But having made your first incision too big, you cannot make it smaller again by stitching it partly up, for your scar will correspond in length to that of the incision. In the third place, in considering the resulting disfigurement, you may remember that sometimes it will be better to make two incisions than to make one long incision. I would point out that, because the glands are affected in the anterior as well as the posterior triangle, it is not in the least necessary that you should make two incisions to get them out; you may often get them all out through a posterior incision. Pass your finger or an instrument below the sterno-mastoid muscle and get the glands out by means of such instruments as I show you here, and do as little dissecting as possible. If the glands are adherent or the surrounding parts are affected you may, of course, be forced to dissect the whole out with a knife. I use one of these elevators, or push an aneurysm needle around the capsule of the gland, and gradually separate it from the surrounding parts until at last the gland is drawn up out of the wound, and the few adhesions which still bind it down are either torn through or divided. For this work these blunt-pointed scissors, curved on the flat, are of great service. The knife should be used as little as possible after the beginning of the operation. Of course you get the glands out, if possible, without breaking them. But you are not able to do that if they have suppurated to any great extent, and then you will have pus in your wound. When that is the case how shall the wound be treated? I do not use much lotion after the operation, but I mop out or rub the cavity very freely with antiseptic gauze or wool. If there has been any pus, or if a cavity is left with resisting walls, I always insert either a fine drainage-tube or tissue, or a piece of sterilised silk, for the first few days. Afterwards if there has been suppuration I have the wound syringed out for the first few days, until the danger of tuberculous suppuration has passed away. In bringing the edges of the wound together I use very fine silk (No. oo) with a very small needle, and I put in interrupted sutures because I think they produce less wrinkling and puckering of the skin. The sutures are taken out not later than three days after the operation, and I have now begun to take them out two days after the operation, because if they are left for any length of time they leave little marks. If you fear that your wound will come apart, put one strip of

Digitized by Google

strapping over the dressing, and that will prevent any such catastrophe. I do not know that there are any other special directions than these.

When I was thinking over the subject and jotting down headings, I thought I would like to see how far my practice is the practice of the present day; whether I was telling you what may be regarded as orthodox teaching, or whether I was telling you things which were rather different from the regular routine. So I looked up several of the later treatises on operative surgery and on general surgery, and I found that excision of tuberculous glands is the treatment of the present day, and I found that the rules which I have laid down for myself are very much those of all surgeons, at all events of those who write books at the present time, with certain modifications. One surgeon, for instance, who enjoys a very good reputation in this town as a scientific surgeon, and who has written a very excellent book, recommends that where peri-adenitis is present in the neck, and where the glands are suppurating, and where they are adherent to the surrounding parts, a very long incision should be made; that the parts should be exposed, and, suppose the glands affected are in the anterior triangle of the neck, that all the contents of that triangle—fat, connective tissue, and the mass of glands—should be removed in one continuous mass, much in the same way as I am in the habit of removing the contents of the anterior triangle in a case of malignant disease, with the exception that one would not think of removing the submaxillary salivary gland for tuberculous disease. And that surgeon goes further and says if the mass of glands is adherent to the jugular vein he thinks it is better to tie the vein above and below the adherent mass, and cut out the intervening portion. Now, of course, if you make an incision from the clavicle to the mastoid process, and you expose the whole of the triangle, as I expose it for the removal of glands in connection with malignant disease, the operation is simple enough, and not so dangerous as when you perform it through a small incision. But with such an incision as that you will have a considerable scar; and the question is whether that is I will not say justifiable, but whether it is necessary. I cannot help thinking it is not necessary. There are men who recommend treatment which they do not themselves perform, and I have seen suggestions in books at which the author has been surprised when they have been pointed out to him. But I understand that this particular author does carry out his own recommendations. I think he must have in mind the infectivity of tubercle, and he must himself feel that if the gland becomes adherent to the jugular vein there is a danger, not merely of other glands becoming infected, but that the tubercle may be carried into other parts of the body, and that the patient may suffer from general tuberculosis. Of course that is a possibility, but I believe it is a possibility so remote that I cannot remember any instance of patients who have been brought to me on account of glandular affections of the neck who have, as a result of that, appeared to become the subjects of general tuberculosis. I should not be very much opposed to this method of treatment if one could depend on removing *all* the tuberculous disease, even by so large an operation. But it is almost out of the question. Look at this girl, here, who has a tuberculous gland in the neck. If you examine the other side you will find she has some enlarged glands there also; and there is scarcely a single instance in which there is extensive disease of the glands in the anterior triangle of one side in which the glands in the posterior triangle on the same side are not affected, and there is frequently affection also of the glands on the opposite side. So, even by this large operation, you do not get rid of the whole of the tubercular disease, and therefore of all the possible sources of infection of the internal organs.

Let me warn you of one thing, not to think lightly of these operations for tuberculous glands. By-and-bye you will have children and young people brought to you with tuberculous glands in the neck, and you may form the idea that the operation is easy, and that it will bring you credit. But as time goes on I think you will come to regard operations for this condition with less and less enthusiasm. I have gradually come to the conclusion that the best thing one can hope to do is to leave the case without a diminution of one's surgical reputation. To get out a large number of glands through a small incision in an inconvenient situation is difficult enough; but how much more difficult when the glands are widely scattered, softening, breaking-down, adherent to, and sometimes incorporated with the surrounding structures, which may be the main nerves or vessels of the neck.

Digitized by Google

I have heard of more than one fatal accident in the course of these operations, and I am surprised that accidents are not more frequent. The worst thing which has yet happened to me has been to have to ligature the common carotid artery, and that was serious enough. Some years ago I was operating upon a little boy in a nursing home, taking out a chain of caseous and suppurating glands—fortunately I had lengthened my incision, because the glands were very adherent, and they lay upon the line of the big vessels of the neck,—when there came a sudden burst of hæmorrhage from the carotid artery. The blood actually spurted to the ceiling, and my assistant was for the moment so startled that he jumped back instead of doing what he should have done to stop the bleeding. I had great difficulty in applying a ligature above and below the point where the coat of the artery had been torn, and I really thought the poor, delicate little fellow might die upon the table. But at length the artery was tied, the glands were removed, and finally the patient made a good recovery.

If a sudden and severe hæmorrhage happens at the bottom of a deep hole, where you are operating through the smallest possible wound, so as not to impair the appearance of the patient in after life, you can imagine what the difficulty would be. In such a case I would recommend you instantly to plug the bottom of the wound with gauze, and then make a much larger opening, and deal with your enemy face to face. It is no longer a question of appearance, it is a question of life and death.

There is another danger which is always present in my mind—the fear of damage to the spinal accessory nerve. You know what the effect of that is, and how little credit it will bring you as a surgeon. To tell the truth, there are many capital operations in surgery which I would rather face than an operation for the removal of tuberculous cervical glands, and I am always pleased when it is over. Still, you will have to face such cases, and you must determine to do the best you can for the patient, with the least thought for your own reputation.

THE importance of the exact dosage of sodium cacodylate has induced Burroughs Wellcome and Co. to issue the drug in tabloid form; purity and accuracy in prescribing it can therefore be assured.

A CLINICAL LECTURE*

ON

A CASE OF MUSCULAR ATROPHY.

By J. H. BRYANT, M.D., F.R.C.P.,

Assistant Physician to Guy's Hospital.

GENTLEMEN,—I propose to-day to demonstrate and to lecture to you on a case of muscular atrophy, at present under my care in Miriam Ward. Mr. Bevers has made a very excellent and careful report, and I will first of all read to you an account of the case based on his notes.

Lilian S—, æt. 9, admitted April 11th, 1901, for wasting of the legs and hands. Her mother states that when she was born forceps had to be used. Although she was a small child she was considered strong and healthy, and could walk at the age of nine months. She then had a bad attack of bronchitis, and to use the mother's words, "was taken off her feet until she was fourteen months old." She suffered several times from bronchitis during the next three years, and when she was four she had an attack of whooping-cough and was ill for three weeks. When she recovered she was unable to walk. Her mother said "that she did not seem to know how to take her steps." On account of this she was taken to the Orthopædic Hospital, in Oxford Street, where a diagnosis of infantile paralysis was made. She was ordered to wear boots with iron supports.

The mother described both feet and legs as being wasted as far up as the knees at this time, the right being more affected than the left, and she also stated that at times the child used to fall down, her knees giving way without any warning. She was quite unable to get about without her boots. When she was five and a half she had scarlet fever, and at six and eight attacks of measles. Fifteen months ago the mother noticed that both the child's hands and body appeared to be getting thinner. At times she complained of aching pain in the feet, and could not walk as far as she used to. On several occasions she suffered from tonsillitis. The mother also stated that the wasting and weakness of the hands and body have been getting worse since she first noticed it. At times she has complained of pain in the right foot, passing up the right side into the right arm, and also of

* Delivered at Guy's Hospital.

Digitized by Google

headache. She has of late also not been able to hold or pick up things as well as other children, and she writes slowly and in a peculiar manner. When doing nothing she allows her hands to hang limply in front of her. During sleep she has been noticed to clench her hands. She has never had a fit, and has always had perfect control of her urine and fæces. She has never had any sign of chorea.

Both her parents appear to be strong healthy people. The father states that one of his brothers suffers from weakness and wasting in his arms and legs, and that he has a sister who has had fits of an epileptic character, and that another of his sisters is afflicted with heart disease. Both his mother and father are well, and with the exception of his brother mentioned above, no history of muscular wasting in any other member of the family can be obtained, and the same applies to his wife's family. He has been the father of nine children, of whom five are living and are all strong and well, with the exception of this little girl. He further states with regard to his brother that he was late in learning to walk, that at the age of three weakness commenced in the feet without any apparent cause, there being no history of any acute illness at this time. Wasting of the muscles followed the weakness, and at the age of ten the hands were affected in the same manner with both weakness and wasting. From this time the trouble has been progressive in character. He is now thirty-nine years of age, and the wasting has slowly extended from the feet as far as the knees, and from the hands to the forearms. At an early date he was able to get about with the help of a stick, later he had to resort to crutches. At the present time he is quite helpless as to his hands and feet, and wears pads on his knees, dragging his feet and legs behind him as he moves along, whilst his hands are so weak that he is quite unable to hold his short crutches, and to remedy this, iron hooks have been attached to his forearms and with these he grasps the crutches. He appears to be strong and healthy in other respects, without wasting of his body, upper arms, or thighs.

You see that she is a healthy, intelligent, bright-looking child. At times she lets her hands fall limply in front of her, and if they are raised from the bed a condition of wrist-drop becomes apparent. There is marked wasting of the thenar and hypothenar muscles of both hands, and also of the interossei. With regard to the forearms, they are neither very large nor very small, perhaps on the whole there is slight wasting. The muscles of the trunk do not appear to be much wasted, if at all. The scapulæ are rather prominent, and there is a slight spinal curvature in the dorsal region, with the convexity backwards and a little lordosis in the lumbar region. She has full use of her arms, and free movement of the hands with a very fair grip. The deltoids and scapular muscles are not wasted, nor are the facial muscles. She holds a pen like other people but writes very slowly. As she lies in bed the feet are extended, there is slight talipes varus, and marked wasting of the muscles of the legs below the knees. There is no wasting of the thigh muscles or of the glutei. None of the muscles are abnormally large or hard, and there is no history of the muscles having previously been large and hard. When she first gets out of bed she cannot walk and can only just stand, but she soon recovers and gets about fairly well. The gait is of a high-stepping character. There is slight genu valgum.

The sensory functions appear to be normal. There is no loss to touch, pain, heat, or cold, and all these forms of sensation can be appreciated and differentiated.

The knee-jerks are absent. There is no ankle clonus, wrist jerk, or elbow-jerk. The plantar reflex is present, and is a flexor response. The abdominal, epigastric, and scapular reflexes are present. The pupils react to light and to accommodation.

The left leg is weaker than the right, but she can lift both legs off the bed against a fair amount of resistance. The movements of the knee and hip-joint are good.

There is well-marked wrist-drop, but she has a fair grip in both hands. The movements of the thumbs are rather weak. There does not appear to be any weakness of the upper-arm muscles or of the shoulder or scapular muscles. There is no spasm, tremor, or inco-ordination. Slight fibrillation has been noticed.

With the faradic current there is no response in the thenar and hypothenar muscles and the interossei, with the exception of the third left dorsal, in which a slight contraction can be obtained.

The flexor and extensor of the wrist respond

Digitized by Google

normally. Also the shoulder, upper-arm, scapular muscles, and the muscles of the face.

The small muscles of the feet—the peronei and anterior tibial muscles—do not react, but the calf muscles, the glutei, and the thigh muscles react normally.

With the galvanic current there is no reaction in all those muscles which do not react to the faradic current, but in the other muscles K.C.C. was greater than A.C.C.

No changes in the optic disc, retina, or choroid.

The circulatory, respiratory, and abdominal systems do not present any abnormal changes.

The urine is acid, light in colour. Sp. gr. 1030, and contains no albumen, sugar, pus, or blood.

The most noticeable feature of this case is the muscular atrophy which has involved the feet, legs, and hands. There are also well-marked changes present in the electrical reactions of the muscles which show most atrophy, for they react neither to faradism nor to galvanism. In first of all considering the diagnosis of this case it is therefore evident that a careful analysis must be made of all the symptoms and signs of the various diseases which are characterised by muscular atrophy, in order to see with which type it coincides.

Muscular atrophy may result from lesions of :—

1. The anterior cornua.
2. The anterior nerve-roots.
3. The nerves.
4. The muscles themselves.

The following is a list of the several diseases :—

1. *Anterior cornua.*
   - (*a*) Acute anterior poliomyelitis.
   - (*b*) Subacute and chronic poliomyelitis.
   - (*c*) Progressive muscular atrophy.
   - (*d*) Amyotrophic lateral sclerosis.
   - (*e*) Diffuse myelitis.
   - (*f*) Hæmatomyelia.
   - (*g*) Syringomyelia.
2. *Anterior nerve-roots.*
   - Compression by caries.
   - Compression by growth.
   - Compression by hæmorrhage.
   - Compression by pachymeningitis.
3. *Nerves.*
   - Injury.
   - Neuritis.
4. *Muscles.*
   - Pseudo-hypertrophic muscular paralysis.
   - Idiopathic muscular atrophy.
     - (*a*) Erb's type.
     - (*b*) Landouzy-Déjerine type.
5. *Neuro-muscular.* Tooth's peroneal type.

A certain amount of muscular atrophy may result from disuse, and may be associated with paralytic conditions due to lesions of the pyramidal fibres above the anterior cornual cells, *e. g.* to lesions of the cortex, corona radiata, internal capsule, crus, pons, medulla, or pyramidal tracts of the cord. The muscular wasting which results from such causes is never accompanied by reaction of degeneration, or by a loss of the electrical reactions such as is found in this case.

Children not infrequently suffer from paraplegia as a result of cortical lesions at birth. It is distinctly stated by the mother that this child could walk when she was nine months old. It is not at all likely therefore that her present condition is in any way connected with an injury to the cortex at the time of the instrumental delivery. The loss of reaction of the muscles to faradism and galvanism and the absence of knee-jerks also are points against a cortical or cerebral lesion.

The failure of certain of the atrophied muscles to react to either faradic or galvanic stimuli has a most important significance, for in addition to excluding lesions of the upper neuron it indicates that the cause of the trouble must be in the anterior cornua, anterior nerve-roots or motor nerves, and practically excludes the possibility of a simple idiopathic muscular atrophy, by which I mean a condition of muscular atrophy due to changes in the muscles alone, the anterior cornua, anterior nerve-roots, and motor nerves being quite free from any pathological changes.

The possibility of infantile paralysis must be carefully gone into, on account of this disease having been diagnosed at the Orthopædic Hospital. The child was taken there soon after the attack of whooping-cough, and the somewhat acute nature of the paralysis and wasting of the legs must have led to this diagnosis. The relation the disease appears to have to the attack of whooping-cough has a most important bearing on the diagnosis, and I shall refer to it again when discussing the peroneal type of muscular atrophy.

Infantile paralysis is an acute disease charac-

Digitized by Google

terised by pyrexia, loss of power, and rapid atrophy of certain muscles. The lesion which produces these symptoms is an acute inflammation of the anterior cornua.

The cause of the atrophy of the muscles is a destruction of the nerve-cells in the anterior cornua, with a resulting degeneration of the corresponding motor nerve-fibres. The onset is usually very acute, and in some cases may be described as sudden. It is accompanied by marked constitutional disturbances, such as pyrexia, vomiting, restlessness, headache, and even convulsions. These symptoms may last for two or three days, but occasionally they are absent altogether. It is essentially a disease of late infancy. Three fifths of the cases which occur in early life are under three years of age. It rarely occurs before six months. Boys and girls suffer equally. It occurs much more frequently in the summer than in the winter. Epidemic forms even have been described in America. Over-exertion, exposure to cold, and falls appear to be exciting causes. Out of 214 cases analysed by Gowers not one was found to have had any definite relation to an acute disease. Severe pain is often a well-marked initial symptom, more particularly in adults and older children. It is often a misleading symptom, and I have seen it more than once lead to a diagnosis of acute rheumatism. A careful examination will show that the joints are not involved. I remember some years ago, when I was attending the medical out-patients, hearing one of my fellow students ask the physician if there was not often a difficulty in distinguishing between infantile paralysis in the early stages and acute rheumatism. The reply was that there should be no difficulty. The very next case, however, which was examined proved to be a child acutely ill with pyrexia and pain in the legs. The child was admitted to the hospital, the diagnosis being ? acute rheumatism, ? infantile paralysis. After being a few days in the ward there was no doubt about the diagnosis, for loss of power and muscular atrophy had supervened, and it was clearly a case of infantile paralysis.

I have attending at the electrical department a child suffering from well-marked infantile paralysis, who was seen on the second day after the onset by a specialist in nervous diseases. She was feverish and in acute pain, and was considered by him to be suffering from rheumatism. A few days later she was seen by an obstetric physician, who, finding considerable loss of power in her legs, was able to make the correct diagnosis of infantile paralysis. A few cases commence less acutely, the loss of power taking two or three weeks to develop. The paralysis is not progressive in this disease, but, on the other hand, rather regressive, as there is a tendency for a certain amount of recovery, except in a particular part or parts where the muscles rapidly waste. Disturbances of sensation, such as anæsthesia, analgesia, etc., are usually absent. The reflexes are lost in the affected parts. In addition to the atrophy of the muscles other trophic changes, such as retardation of the growth of bone, may be noticed. Later on there is a tendency to permanent shortening of the muscles, a condition which may lead to displacement and deformities. The sphincters are very seldom involved. The muscles supplied by the cranial nerves are very rarely affected.

Gowers divides the disease into four stages :—

1. An initial stage of paralysis accompanied by fever, lasting from a few hours to a week or more.

2. A stationary period, lasting for a week to a month.

3. A stage of "regression," during which the paralysis passes away, except in the parts where wasting occurs, the period lasting from one to six months.

4. A chronic stage, during which the atrophy continues, and slight improvement occurs, but deformities develop.

I think you will be able to see from what I have told you about infantile paralysis that the little girl I have just shown you is not suffering from this disease. There are several important points against such a diagnosis. First of all there is the absence of a history of any acute onset, then there is the distribution of the loss of power and atrophy —it is extremely rare to get both feet and legs and both hands affected as the result of infantile paralysis. Then there is the relation of this disease to the attack of whooping-cough. I have already drawn your attention to the fact that in 214 cases of infantile paralysis collected by Gowers not a single case can be traced to the effects of an acute disease. Then there is the fact that the disease has definitely been progressive, the hands becoming affected several years after the feet and

Digitized by Google

legs. This progressive nature of the disease is a very strong argument against a diagnosis of infantile paralysis, which is essentially a regressive disease.

Before passing on to the consideration of the causes of muscular atrophy I will show you a well-marked case of infantile paralysis, and compare it with the case you have already seen. The following is a short history :—Winifred W—, æt. 4½, was brought to the electrical department for loss of power and wasting of the right arm. On July 30th, 1900, she was running and fell suddenly on her right side. She tried to save herself by putting out her right hand. On the following day the right arm was found to be powerless, and she was very feverish and ill. Wasting of the muscles soon followed. She never had much pain in the arm, and there was never any loss of sensation. She was treated at the Children's Hospital, Great Ormond Street, for some time. She came to the electrical department on January 30th. There was marked atrophy of the deltoid, biceps, and triceps, and of the muscles of the forearm and hand. There was no loss of sensation. There was well-marked reaction of degeneration. The condition now is practically the same.

A few cases of muscular atrophy are due to subacute or chronic polio-myelitis. They are characterised by a much less rapid onset. Paralysis followed by atrophy of the muscles comes on gradually, if within thirty days they may be termed subacute, and if from one to six months chronic.

Cases of this kind are difficult to differentiate from chronic myelitis and from multiple neuritis. They occur most frequently in adults. The long period between the occurrence of the changes in the hands and the onset of the disease, with weakening and wasting of the muscles of the foot and leg, and the age of the patient are, I consider, the important points against the disease being of this particular nature.

The marked wasting of the thenar and hypothenar muscles is one of the changes which is first noticed in cases of progressive muscular atrophy. This is a chronic disease which is due to a degeneration of the anterior cornua of the spinal cord. The degeneration involves the ganglion cells, and is accompanied by a degeneration of a similar character in the motor nerve-fibres which come from the affected cells. Associated with this degeneration there is usually also a degeneration of the pyramidal tracts of the cord. Gowers writes, "I have not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts are unaffected." He does not distinguish between progressive muscular atrophy and amyotrophic lateral sclerosis. As progressive muscular atrophy and amyotrophic lateral sclerosis are described as two different diseases in Dr. Taylor's 'Medicine,' the text-book you all should rely on, I shall, in order not to confuse you, discuss these two conditions separately.

Progressive muscular atrophy is a degenerative disease of the anterior cornua, first involving the lower cervical and upper dorsal regions of the spinal cord. It occurs more frequently in males than in females, and it usually commences between the ages of twenty-five and forty-five. The youngest case recorded, that I know of, is one in which the disease began at twelve.

Weakness and wasting usually come on together, and the onset is insidious and gradual. Often no cause can be traced. The thenar and hypothenar muscles are usually first affected, and they may be followed by weakness and wasting of the muscles of the arm and shoulder. Fibrillary contractions are usually well marked. Faradic and galvanic excitability are usually both diminished, and there may be a partial form of reaction of degeneration. The reflexes are lost. There is no anæsthesia, and the bladder and rectal functions remain intact.

The age of the patient is a very strong argument against the disease being progressive muscular atrophy, for she has not yet reached the age of the youngest case recorded. The commencement of the weakness in the feet and legs with the loss of knee-jerk are other points of importance against this view.

Amyotrophic lateral sclerosis is a disease characterised by a degeneration of the anterior cornual cells and by sclerosis of the crossed pyramidal tracts. The anterior cornual cells are involved in the lower cervical and upper dorsal regions of the cord, and the crossed pyramidal tracts are involved as a rule throughout the cord. The disease usually begins in the hands and arms with wasting of the thenar and hypothenar muscles; fibrillary muscular contractions occur, and the electrical reactions are similar to those obtained in progressive muscular atrophy. In addition to these changes

Digitized by Google

in the upper extremities there are the symptoms produced by the lateral sclerosis, viz. loss of power in the legs, with rigidity, exaggerated knee-jerks, ankle-clonus, and extensor plantar response (Babinski's sign).

It is evident therefore that the little girl is not suffering from this disease. She is still too young, the disease in her case commenced in the legs, and the arms and legs were only affected several years afterwards; and further, the condition of her feet and legs are obviously not due to a lesion of the crossed pyramidal tracts, for her knee-jerks are lost, there is no ankle clonus, no extensor plantar response, and no rigidity; and then, there are the altered electrical reactions.

The mode of onset, the absence of sensory disturbances, the normal condition of the bladder and rectal functions exclude the possibility of her condition having been due to a diffuse myelitis, for that disease is characterised by its acute onset, with tingling and numbness, followed by a rapidly developing paralysis. All four limbs become paralysed, there is general anæsthesia, the muscles waste, the reflexes are lost, and there is retention of urine and involuntary passage of fæces. Death as a rule soon follows.

Hæmatomyelia or hæmorrhage into the spinal cord may be followed by muscular atrophy. It is distinguished from other diseases by the very sudden onset. The symptoms resemble those produced by acute myelitis. It would be quite impossible for hæmatomyelia to have produced such changes as you have seen in this little girl without also causing very marked sensory, trophic, and visceral changes.

Syringomyelia is another disease which may be accompanied by marked muscular atrophy. The term syringomyelia is used to denote a condition of cavitation of the spinal cord. The cavities are often congenital, and they may be associated with a diffuse gliomatous condition. The two chief symptoms of this disease are loss of sensibility to pain and temperature and muscular atrophy, which may resemble in its characteristics progressive muscular atrophy. The muscular atrophy is a result of the damage to the anterior cornua from the compression of the cavity and its adjacent gliomatous growth. The motor nerve-cells are damaged or destroyed, the corresponding nerves degenerate, and the muscles they supply, atrophy. The absence of the characteristic sensory disturbances, viz. the loss of sensibility to pain and temperature, make it, I think, quite clear that she is not suffering from syringomyelia.

Muscular atrophy may also be a result of diseases of the anterior nerve-roots. They are not usually primarily affected, but may be pressed on as a result of caries or growth of the spine, by meningeal hæmorrhage, and by thickening of the meninges. In the case of caries and growth changes may be noted in the spine. Meningeal hæmorrhage comes on quite suddenly. They are all, however, characterised by shooting pains in the back, neck, and arms. Anæsthesia, loss of reflex, and reaction of degeneration may also result.

There is no evidence to even suggest that the anterior nerve-roots are involved in the case before us.

There is no history of injury, and it would be most unlikely for any injury to pick out so many nerves as would correspond with the distribution of the weakness and atrophy.

The possibility of a multiple neuritis being the cause of the atrophy must be much more seriously discussed. The fact that all four limbs are affected, the curious high-stepping gait, the wasting of the muscles, the changes in the electrical reactions, the onset after an acute disease are all conditions which may be explained by a multiple neuritis.

Against a diagnosis of multiple neuritis is the curious way in which the disease has progressed. It is quite unlike a multiple neuritis to produce weakness and wasting of the legs at the age of three, and weakness and wasting of the muscles of the hands in the same patient between seven and eight, without any apparent cause or reason; and, further, no cause for a multiple neuritis could be traced. In children a multiple neuritis is most likely to be the result of diphtheria. There was no evidence at all of the child ever having suffered from it, and further there was no history of fluid regurgitating through the nose, of nasal voice, or of squint, all conditions which are very commonly found in patients suffering from diphtheritic neuritis. The atrophy of the thenar, hypothenar, and interossei might suggest the possibility of plumbism, but there has not been, and there is no collateral evidence of such a condition. I consider therefore that we have not sufficient evidence to

Digitized by Google

say the muscular atrophy is due to a simple multiple neuritis.

I must now pass on to the consideration of the various forms of primary or idiopathic muscular atrophies, diseases which are characterised by primary changes in the muscles themselves, the nerves and spinal cord being normal. The occurrence of a simple affection in another member of the family and the early onset of the disease are two very important diagnostic indications of some form of idiopathic muscular atrophy. A good deal of confusion appears to exist in the description of the different forms of idiopathic muscular atrophy.

I propose to consider them under four headings :—

1. Pseudo-hypertrophic muscular paralysis (Duchenne's type).
2. Juvenile muscular atrophy (Erb's type).
3. The facio-scapulo humeral type (Landouzy-Déjerine type).
4. Mixed and intermediate forms of the above.

Erb has carefully examined a large number of clinical records of these forms, and has come to the conclusion that they are all varieties of the same disease. One very important point to remember is that hypertrophied muscle may be found in certain parts of the body in all these types.

As to the nature of these curious forms of muscular atrophy, I cannot do better than quote Gowers, "All the diseases of this class seem to depend on a defective tendency in the development of the germinal tissue which forms muscles ; they are essentially congenital diseases."

Pseudo-hypertrophic muscular paralysis is a disease characterised by enlargement of the muscles with marked loss of power. The hypertrophied muscles subsequently atrophy, and in some cases atrophy occurs in muscles which have never passed through a stage of enlargement. The muscles are usually hard, and feel quite different to the soft, flabby, atrophied muscles which result from a lesion of the anterior cornua or the nerves. The characteristic change in the muscle is a deposit of fat and fibrous tissue between the muscle-fibres with subsequent atrophy of the fibres. It affects boys more frequently than girls. Several members of the same family are often affected. It is usually transmitted through the mother. The disease as a rule manifests itself when the child is about five or six years old, or when it first begins to walk. The gait may be unsteady and waddling in character. The calf muscles may be large and hard. If carefully examined for, in addition to these hypertrophied muscles, others may be found showing marked atrophic changes. The most characteristic features of the disease are the curious waddling gait, the feet being wide apart and the body swaying from side to side. The tendency to walk on the toes, the marked lordosis, the peculiar method of rising from the prone to the erect position (here is a photograph illustrating this point), and the association of enlargement of the infraspinatus with atrophy of the lower part of the latissimus dorsi and the pectoralis major.

There is no evidence of the case under consideration being one of pseudo-hypertrophic muscular paralysis. There is no history of any of the muscles ever having been hypertrophied, and there is no evidence of any of the muscles being hypertrophied or unusually hard at the present time.

The child has not the gait which is characteristic, nor does she, if laid down, rise from the floor in the manner which is more or less typical of pseudo-hypertrophic muscular paralysis. One of the most important diagnostic signs of pseudo-hypertrophic muscular paralysis is enlargement of the infraspinatus combined with wasting of the lower part of the latissimus dorsi and pectoralis major. There is certainly no such hypertrophy or atrophy in this case.

In the juvenile or Erb's type of idiopathic muscular atrophy, wasting and weakness usually come on simultaneously in the upper arm and shoulder muscles. The biceps, triceps, and supinator longus suffer. The lower part of the latissimus and pectoralis major may be also wasted. The forearm muscles usually escape, and in the majority of the cases the thenar, hypothenar, and interossei muscles are unaffected.

In the Landouzy-Déjerine type the facial muscles are involved. The zygomatic muscles waste, and there is a loss of the naso-labial furrow. The orbicularis oris is affected and, as a result, the lips are continually separated. The expression is very dull, and the face has been called by Landouzy and Déjerine the "myopathic face."

A further effect of the wasting of these facial

Digitized by Google

muscles is a curious alteration in the smile, the angles of the mouth being drawn upwards instead of upwards and outwards. The patients cannot whistle and cannot perfectly articulate labials. In addition to the humeral and scapular muscles being affected, the abdominal, spinal, flexors of the hip, and the extensors of the knees may become involved. Fibrillation is nearly always absent. The electric irritability is usually less to both currents. There is no loss of sensation and the sphincters are unaffected. As the atrophy progresses deformities result. The course and duration varies exceedingly. Patients may live for many years, the duration may be said to vary from ten to fifty years. You will see that the case I am discussing does not fit in with any of these types. There is no change whatever in the facial muscles, so that she is not suffering from the Landouzy-Déjerine type. The upper arms are not affected, and the thenar and hypothenar muscles are; there are no hypertrophied muscles; there are very marked alterations in the electrical reactions, some of the muscles not contracting to either faradic or galvanic stimuli. I consider these data sufficient to say she is not an example of Erb's juvenile type of muscular atrophy, of the Landouzy-Déjerine form, or of Duchenne's type.

It is evident from what I have already said, that this case of muscular atrophy I have presented to you does not fit in with any of the types I have discussed with you so far. There, however, remains a rare form of muscular atrophy which was first of all described by Charcot and Marie in 1886, and in the same year by Dr. Howard Tooth in a thesis entitled, "The Peroneal Type of Progressive Muscular Atrophy." It is usually spoken of as Tooth's peroneal type of muscular atrophy. Gowers describes it as "The peroneal type of family amyotrophy" (neuritic muscular atrophy). I consider the little girl I have shown you to-day to be suffering from this form of muscular atrophy.

In some respects the disease resembles the idiopathic muscular atrophies I have just discussed with you, and is therefore, as a rule, described with them. It resembles the primary myopathies in several points—the age at which the disease commences, the gradual onset, the slowly progressive character of the malady, and the occurrence of the disease in other members of the same family. It differs, however, markedly in the distribution of the atrophy, and the very definite relation the onset often has to some acute disease, such as measles, and in the case before you, whooping-cough.

Males suffer from this form of atrophy about twice as frequently as females. It very rarely commences after twenty, but is usually first noticed in early childhood. It is sometimes hereditary. The wasting first appears in the extensor longus hallucis, extensor communis digitorum, or the peronei muscles, and very probably, according to Gowers, it starts first of all in the small muscles of the foot, for a wasting of such small muscles may very easily escape observation. Later on the calf muscles suffer, and still later the vastus internus. Talipes often results on account of the unequal manner in which the muscles of the legs are affected. Some years after the legs have been implicated the hands and forearms are involved, the thenar, hypothenar, and interossei muscles being first of all attacked. The supinator longus usually remains normal, and so also do the muscles of the shoulder, back, and neck. The atrophy is usually, but not always, symmetrical. In advanced cases a claw-like deformity of the fingers may result from the atrophy of the intrinsic muscles of the hands. When this deformity is associated in early life with the wasting of the muscles of the feet and of the peronei, etc., it is almost diagnostic of this peculiar type of muscular atrophy. Occasionally slight fibrillation of the affected muscle is observed. Another very important change is the diminution of the electrical irritability, a point which is of great importance in distinguishing cases of this nature from those of idiopathic muscular atrophy. Faradic irritability early becomes lost, and there may be well-marked reaction of degeneration. Cutaneous sensibility is usually preserved, but it may be lost or impaired. Pains and spasmodic contractions of muscles are noticed in some cases. The knee-jerks may be lost. The cutaneous reflexes vary, and are often not lost.

You will see that in most respects the case under consideration fits in with this type of muscular atrophy. First of all there is the age of onset—loss of power in the legs was first noticed when she was four years old, and it followed an attack of whooping-cough.

The family history is of importance, for one of

Digitized by Google

the uncles appears to be suffering from a disease of a similar nature, which commenced when he was three years old, and it has been gradually progressing ever since. The distribution of the atrophy, and the mode of onset and progression are quite characteristic, for it first of all commenced in the feet and legs, and three years afterwards attacked the intrinsic muscles of the hands. The muscles of the face are not affected, nor are the shoulder, neck, or back muscles. The marked diminution of electrical irritability is another important point of similarity, and so also are the attacks of pain in the feet from which she occasionally suffers, these last two conditions being important diagnostic points of distinction from true idiopathic muscular atrophy.

Then as to the nature of this form of muscular atrophy. I have already alluded to its similarity to the idiopathic muscular atrophies in its slow and progressive course, the age at which it begins, and the affection of other members of the same family, and also to its dissimilarity in that it is so prone to follow an acute specific disease. This last-mentioned important factor in its causation, the alteration in the electrical reactions, and the occasional presence of anæsthesia are all features which point to marked changes in the nerves as a cause of the disease. There are very few cases in which a thorough post-mortem investigation has been made.

Dubreuilh has examined a case and found inflammatory changes in the nerves, the spinal cord being quite free. Tooth advocated a degenerative form of neuritis as the true pathology, and pointed out that such a view would help to explain the occurrence of fibrillation, which is of extremely rare occurrence in any of the idiopathic muscular atrophies. It is, of course, possible that there is a liability to early slow degeneration in the nerves as well as in the muscles. It would appear that in some cases the muscles atrophy because of degeneration of the nerves alone, whereas in others the muscles have a congenital tendency to atrophy as well as the nerves to become degenerated. If there is such a congenital tendency to degeneration it is easy to understand how a disease like measles or whooping-cough may act as an excitant and start the process. The slow nature of the process and the liability of other members of the family to be affected serves to distinguish it from cases of pure neuritis.

The prognosis is by no means good. The tendency of the disease is to gradually but surely progress. I am afraid the future of the little patient is foreshadowed by the present condition of her uncle, who now is thirty-nine years of age, and has the greatest difficulty in getting about, having completely lost the use of his hands and feet. Patients rarely die of the disease itself, but usually succumb to some intercurrent affection, such as pneumonia, tuberculosis, etc.

Very little can be done in the way of treatment. No good can be hoped for from electrical treatment or massage. The general health must be attended to, over-exertion avoided, and strychnine administered internally.

CLINICAL REMARKS ON FOUR CASES OF INTRA-OCULAR HÆMORRHAGE.

By W. M. BEAUMONT, M.R.C.S.,
Surgeon to the Bath Eye Infirmary.

DIVERSE and many are the causes of intra-ocular hæmorrhage. These four patients justify the first adjective. Here is a man æt. 27 with nothing particular in his family history, whilst his personal story is perhaps more important than it appears to be at first sight. He is subject, he tells us, to headaches and epistaxis, the latter having occurred at intervals of about a month for many years. He does not bleed excessively when he gets an accidental scratch or cut. Three days ago he suddenly lost sight in the left eye; he awoke and was blind on that side. On examining the eye to-day we find that it will not light up, no ray from the ophthalmoscope can pierce the darkness, and it may be said of it, as has been said of amaurosis, that the patient sees nothing and the surgeon sees nothing. Hæmorrhage has occurred into the vitreous, and not a glimmer of light can traverse the cloudy media. On questioning the patient he tells us that six years ago a similar event happened, and that after a few weeks vision gradually returned, and he saw as well as ever. Moreover, he says that twelve months ago a second attack occurred, which again passed off in a few weeks. (A week after this man was first seen a red, sanguinary reflex could be made out by oblique illumination, and gradually a clearing went on until floating

Digitized by Google

streaks of blood-clot could be seen in the vitreous, and finally, in thirty-one days from the onset, perfect absorption and restitution with vision = $\frac{6}{6}$.) This patients belongs to a class, not very common in ophthalmic practice, in which the classical symptoms are headache, constipation, and nose-bleeding, usually occurring in young men. In this case the constipation was not very marked. They have been called cases of "ocular epistaxis," the blood coming from the retinal veins. They do not usually occur in young women, in whom menstruation is supposed to act as nature's overflow.

Our next patient is a woman æt. 48. Her menstrual periods were quite regular until three months ago when, after missing three periods, she awoke one morning and found that she was blind in the right eye. Now, although this occurred a week or two ago, she can only just locate the window and see shadows. She has never suffered from amenorrhœa before, and she, probably correctly, assumes that the blindness is due to the "change of life." In her case the prognosis is more serious than in that of the young man. In the young it is remarkable how rapid and complete are absorption and restitution, whilst in the more advanced in years they are slow and incomplete. In young men, I have said, these cases are sometimes called "ocular epistaxis," in women they may be called "ocular menstruation."

Let us turn now to a third case. This child, æt. 14 months, was brought to the out-patient room with a history of right-eye disease, of an unknown nature, since birth. The globe is greatly enlarged, it is proptosed, and the cornea is quite opaque. The tension is increased and there is evidently pain. On enucleating the eye we find the vitreous full of blood-clot together with some recent blood. The eye is quite disorganised, and it contains a new growth which the microscope shows to be of a gliomatous nature. The eye should have been removed at a much earlier date; at this late day the prognosis is most grave, for the growth will most probably recur in the liver or elsewhere.

The fourth case to which I would refer is that of an aged gentleman whose lens I extracted for cataract. All went well for a day or two when in a fit of temper with his wife he tore off his bandages and threw them across the room. The very natural result was intra-ocular hæmorrhage. I was sent for at once and, on arriving, found the vitreous and blood escaping from the corneal operation wound. The eye, hopelessly lost as a seeing organ, points two valuable morals—first, that a patient who has had a cataract operation should postpone quarrelling with his wife as long as circumstances will permit; and secondly, that all cataract patients should be nursed by a trained nurse. In this particular instance the saving of expense was a very important matter, and having no reason to suppose that beneath his benevolent-looking face there lurked a diabolical temper, contrary to my usual custom, I allowed him to be nursed by his wife.

These four cases illustrate the statement I began with, namely that diverse and many are the causes of intra-ocular hæmorrhage. Amongst the other common causes may be mentioned blows and anything which reduces the tension of the eye. For treatment, local applications of cold may relieve pain if it be present, and may prevent the further effusion of blood. Firm bandaging may also be applied; the recumbent attitude and absolute rest of body and mind should be insisted on, stimulants should be restricted and straining avoided. General treatment by large doses of iodides are worse than useless, but if kidney or heart disease be present appropriate treatment for them should not be omitted.

Rheumatism, Rheumatoid Arthritis, and Subcutaneous Nodules.—Dr. C. O. Hawthorne has just written an interesting book on these subjects, published by Messrs. J. and A. Churchill. Dr. Hawthorne's object is to show that on the most material points concerning the relationship which exists between the various events that are more or less commonly designated "rheumatic," no confident conclusion has yet been reached. As the author explains, it is not yet possible to arrive at any dogmatic declaration in regard to the different phases of rheumatism, in consequence of the apparent contradictions which any enquiry into the facts of the case discloses. The writer has dealt with his subject accurately and concisely, and is to be heartily congratulated on having expressed his views in such admirably clear language, and on having included everything of real importance. The work is one that may be recommended to all interested in the advance of medicine, and the more so because Dr. Hawthorne's teaching is as reliable as it is instructive.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 464. WEDNESDAY, SEPTEMBER 18, 1901. Vol. XVIII. No. 22.

CONTENTS.

PAGE

*A Clinical Lecture on Strabismus. By PRIESTLEY SMITH, F.R.C.S. 337

*Gonorrhœa in Women. A CLINICAL LECTURE. By T. W. EDEN, M.D. ... 342

*Two Clinical Lectures on the Control of Arterial Hæmorrhage and the Treatment of Aneurysm. By W. MCADAM ECCLES, M.S., F.R.C.S. LECTURE I 348

Bone Grafting 352

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A CLINICAL LECTURE ON STRABISMUS.

Delivered at the Medical Graduates' College and Polyclinic, July 10th, 1901.

By PRIESTLEY SMITH, F.R.C.S.

GENTLEMEN,—I wish to speak of a very common disorder—the strabismus or squint which we see so frequently in children. The treatment of squint falls mostly, and I suppose will fall more and more, into the hands of ophthalmic surgeons, but every medical man meets with these cases from time to time, and is asked to advise. Commonly you see them before we do. It is, therefore, very desirable that every medical man should have a clear idea as to the nature of the disorder, and as to the kind of treatment which is required. The special point I want to urge is that at the present time harm is done in many of these cases by delay of treatment. The advice is too often given: "Do nothing at present; wait a few years, and then, if necessary, have an operation." This is usually bad advice.

First as to the nature of the disorder. I cannot show you cases, but we can do something with the model that I have put upon the easel. It shows, in addition to the muscles and nerves, the nerve centres, which, when they act, produce those co-ordinations of eye movements upon which all accurate vision depends. For instance, there is a centre which, when it sends forth its impulse, causes the two internal recti to contract, and to produce the compound movement of convergence. On each side is a centre which sends impulses to the internal rectus of the one eye, and the external rectus of the other, so as to turn both eyes to one side. Let us put the model so that it will imitate a squinting child, the right eye looking straight in front, the left eye squinting considerably inwards. If we show such a child to a class of students who have done no eye work, as I often do, and ask what explanation they can give of it, one or other of them will say, "I suppose there is a fault in one of the muscles."

Digitized by Google

Another, perhaps, will say, "It is a paralysis of one of the nerves." These are good suggestions, but they are not correct. If you move your finger in front of the child's face, and tell him to watch it, his good eye will follow it, and at the same time his squinting eye will move through exactly the same angle. There is therefore no paralysis of nerve or muscle. The real condition of affairs in such a case is an excess of convergence. The child, we will say, wants to look at an object six feet away, but he converges as though for a point one foot away. But he would not look at the point he wants to look at with either eye unless he did something more; so by means of his conjugate centres he turns his right eye straight, and makes the other eye squint double. The nature of the disorder then is this: there is no paralysis, and no fault of the muscle, but the convergent centre acts more than it should, and the conjugate centre rectifies the deviation of one eye, and doubles that of the other. If the child has two equally good eyes, he will sometimes use the right and squint with the left, and sometimes the other way about. If one eye is better than the other, he will generally use the better and squint with the worse.

Why does the convergence centre over-act? The best known cause is hypermetropia. Let us put away all theories and hypotheses, and look at the facts. Examine the refraction of the child before us. Put some atropine in the eyes, and wait from half to three quarters of an hour, and then put the child in the dark room and examine him with the shadow test. Nine chances to one we shall find that he is hypermetropic. Putting it in another way, if we examine a group of squinting children, say 100, with the shadow test after applying atropine, we shall find that at least ninety of them are decidedly hypermetropic, and that they therefore require glasses. You may say, "Well, they may require glasses, but that does not show that the hypermetropia has anything to do with the squint." True, but we can easily prove that it has. Put the ninety children into spectacles which correct the hypermetropia, and look at them again in a week or two. You will find that nearly all the ninety are better; that the squint is less pronounced than before; and that in a considerable number it is actually gone, proving that the hypermetropia was, to some extent at least, the cause of the squint. That is the first point to bear in mind. If a child squints and is not in spectacles, the strong probability is that he ought to be.

Now for the explanation. The hypermetrope, whether he looks at a distant or at a near object, must make a greater effort of accommodation than should be the case. Accommodation is associated with convergence. When we look at a near point we converge the eyes upon it, and accommodate the eyes for that distance. The two efforts are always made together; the centres which preside over them lie close together in the brain; it is difficult to make the one effort without the other. Hence these children who have to accommodate in excess tend to converge in excess. You will say, "That sounds very well, but why do not all hypermetropes squint? There must be something more in it." As a rule a hypermetropic child learns to control these efforts in the way which is necessary for his peculiar condition; that is to say, he learns to accommodate in excess, while he converges normally; he manages to keep his eyes straight, although he has to accommodate too much. Those who cannot learn and maintain this habit have to squint.

Still we want to know why it is that certain of these hypermetropic children squint, while others do not. Why is it that of a family of hypermetropes some squint and others do not? Can we get any further? If you question and cross-question the mother, as I have done in a large number of these cases, and accurately tabulate the results, you will find that in a large number of cases the squint came on during an attack of whooping-cough, or during or after measles or scarlet fever, or something of that kind, or in connection with convulsions, or a slight accident, or something which upset the child's nervous balance. I strongly urge you not to disregard such statements if the facts are clearly stated, because I am sure that in a large number of cases they are true. If we disregard them, and think only of the hypermetropia and the glasses, we shall have only half the truth. I can give you many instances, but I will only mention one or two which have been accurately ascertained, and are sufficient for my purpose. A child goes to school for a year, and gets on pretty well, and has never been seen to squint. One day she mis-

Digitized by Google

behaves, is put in the corner with a cloth over her head, is ashamed and frightened, and comes home with a bad squint. Another child was shut up in a dark room, climbed out of the window—a dangerous climb which she would not have attempted had she not been terrified—was immensely upset, and from that day forward squinted. Another small urchin sat down in a tub of scalding water, and was seriously frightened; she began to squint at once, though she had never been seen to squint before. A sudden shock to the nervous system will bring on a squint; it is natural that it should. Anything which upsets the nervous balance paralyses the higher mental control.

There are other causes which I will not say much about. If you bandage a child's eye, because it is inflamed, for two or three weeks, and he happens to be hypermetropic, you are apt to find when you take the bandage off that a squint has developed. While the eye was covered there was no inducement for the child to keep it straight. Again, there are congenital faults in the nervous system of which we do not know much, maldevelopment of one kind or another, and in many cases the fault is hereditary.

We have also to notice that in the large majority of cases strabismus begins in very early life. During the last few years I have taken a good deal of trouble to tabulate a large number accurately, and I have found that more squints begin between three and four years of age than in any other year of life; that is to say, in children who are not yet four, but who are turned three. Some cases begin under twelve months, some at one year, some at two years, many at three and four years. At five and six years of age the number is much smaller, and for squint to begin at seven or eight is quite uncommon. We see, then, that squint comes on at a time when the visual apparatus is not yet fully developed, when its functions are not firmly established, and when, therefore, they are easily upset. So much for the way in which it comes on.

Now I want to point out, as showing the importance of seeing these cases early, that mischief arises and increases if the squint is not attended to. Let us again look at the facts. Let us take these supposed 100 children and examine them further, especially as regards the condition of the squinting eye. Take the child into a dark room, put a pad over the good eye, throw the light of the ophthalmoscope upon the squinting eye and observe whether it looks at the mirror or not. The way to tell whether an eye is "fixing" or not is to notice the corneal reflex of the ophthalmoscope. Disregard the pupil and fundus and simply look for the bright spark of light which is the reflex of the ophthalmoscope. Anyone can do it. If the eye is looking at the mirror, while you are looking through the mirror, you will see the spark not quite at the centre of the pupil but slightly to the inner side. Why is it not in the middle? Because we do not look through the middle of our pupils. The corneal reflex, then, will tell you whether the eye is looking straight at you or not.

When you cover the good eye and throw the light on the squinting eye, you will see in the majority of cases that this latter is now straight; it fixes the mirror; the corneal reflex is in the right position. You know, therefore, that the child *can* fix with the squinting eye, though generally he does not. On the other hand, a certain number of these patients cannot fix. You will see the corneal reflex displaced outwards, and you will know that the eye is still squinting inwards. And if you come round so as to get it into the right position, the child will move his eye further. That child *cannot* fix the light. I want to insist on the fact that the child does not squint because he has no fixation power, but that he has lost the power of fixing because he squints. Some authorities say that the lack of fixation power is born with the patient. This is true of a few cases where there is cataract or other disease of the eye. It is not true of the large majority of squinters.

If you examine a great number of uncomplicated squints you will never find this loss of fixing power unless the child has squinted at least six months. I found one or two instances where the child had squinted only twelve months, but I found many more in children who had squinted for three or four years. The children who lose the fixing power are those who begin to squint earliest and are neglected the longest.

I shall not trouble you with many statistics, but I will mention this one striking fact: in a group of children, all of whom began to squint before two years of age, and all of whom had been untreated for three years, 87 per cent. had lost fixation power;

Digitized by Google

whereas in children who began squinting later, and were treated sooner, the proportion was much smaller, until among those who did not begin to squint until they were five years old, no loss of fixation-power was found. The reason is plain. The faculty of vision is developed gradually. At birth the child has only a perception of light. During the next few weeks or months he gradually learns to fix with both eyes, and to combine the two images in his brain. But at this time of life the process is easily disturbed, fixation-power is easily unlearned, and learned again in a perverted form.

There are other defects. Many of the children who fix have defective vision in the squinting eye. If a child begins to squint when he is very young the vision of the squinting eye probably never reaches the development which it would have reached under normal conditions. Again, children who sometimes squint with one eye and sometimes with the other may see perfectly well with each eye alone, but if they go on in this way for some years, it is impossible then for them to learn to see with both eyes at once, even though they are put straight. It is clear that a young child who is beginning to squint is likely to suffer detriment if we do not interfere. How can we interfere at this early age?

Let us take the case of children under four years of age, for the majority of squints begin under that age. The question will be put to every one of you now and then: Does my child squint, or does it not? It is most amusing what differences of opinion one hears. The mother thinks there is a squint, the father thinks it is all rubbish, the grandparents are divided on the matter. They go to the doctor to settle it. In such a case you cannot tell whether the child squints by looking at it in the ordinary way. You must look at the corneal reflex in the two eyes, and this is easy, for the youngest child will look at the bright light from the ophthalmoscope. If you see the reflex in the same place in each eye, that is, symmetrically placed, there is no squint. At any rate, there is no squint at that moment. Some children who do not squint appear to do so because of a peculiar arrangement of the fold of skin at the inner angle of the eye; it hangs over so as to almost hide the canthus.

I will not go through all the steps of the examination. When you have ascertained that it is a case of real squint, next test the refraction. Put atropine into both eyes, and keep the child for an hour or two, or let the mother use atropine at home for a day or two; then use the shadow test. The angle of a squint may be easily measured by a graduated tape, but that is a technical matter which it is hardly worth troubling about unless you are practising specially. Further, you can make out whether the squinting eye has or has not the power of fixation. If this is lost, you have a case which is difficult to cure. If the child can look straight at you with the squinting eye when the other is covered, it will be easier to cure. You can then make out whether he always squints with one eye, or whether the squint alternates between the two.

Having made out the various points, what shall we do? The first step in the treatment is almost always to order glasses which will correct the hypermetropia. Of course, if the child has only an insignificant hypermetropia, you would not order glasses, but if he has 2 D. of hypermetropia or more, glasses must be procured. The glasses must fit. A common reason why glasses do not succeed is that they fit badly; they rest too low on the face, and the child looks over them. If necessary they can be made round. They must have hooks behind the ears, and a piece of tortoiseshell should line the bridge to prevent its cutting the nose. Attention to these small matters may make a failure into a success. Children even under two years of age who require spectacles can be taught with little difficulty to wear them. There is also less difficulty with parents in that respect now than there used to be. Years ago it was a difficult thing to persuade parents that a child so young *could* want spectacles. Now they expect to have them ordered, and even expect too much from them. Spectacles cannot pull an eye straight. What they can do is to lessen the effort of accommodation and sharpen vision. A child who only squints occasionally will probably be cured by glasses. If the squint is already permanent, it will almost certainly be diminished by glasses, and in some cases it will be cured altogether.

The second step, which is not much less important, but which is much less practised, is the putting of a pad or shade over the good eye in order to make the child use the squinting eye. It is not easy in a young child with a will

Digitized by Google

of its own to keep the good eye covered, and to make him use the eye which he does not want to use. If you use a hollow shade fastened by elastic he will manage to see underneath it, or by the side of it if there be any gap left. A better plan is to put a pad of Gamgee tissue between the closed eyelid and the spectacles. That makes an excellent shutter. In a few cases the closure sets up a little conjunctivitis, but that does not happen once in twenty cases. At first the child will pull it off perhaps twenty times a day, and the mother will have to replace it twenty-one times, but in a very few days the child gets accustomed to it, and will learn in that way to use the squinting eye. The essential point in getting the pad worn is to make the mother understand its use. We must explain that the child does not use the squinting eye; that he will never learn to use it unless we make him do so; and that he must be taught to use it now or never. Later on we cannot teach him to do so. The value of this treatment is beyond question. I have carried it out in a considerable number of cases, and can speak positively. Even eyes which have no power of fixation at the first visit will sometimes recover it after a few weeks in this way. This I have seen over and over again, but it will not happen if the squint has been in existence for several years. Again, many eyes which have not lost the power of fixation, but which habitually squint, are greatly improved by compulsory use. At the first visit, the good eye being covered, I often put my finger out and say, "Put your finger on mine;" he goes far to one side of it. Or I ask him to put his finger through the ring of my watch; he misses that. But in a few weeks' time when I try him again he goes straight for it. The mother will tell you the same thing; she will say, "We had a lot of trouble with him at first, but now he manages as well with the bad eye as with the good one." I wish to insist on the value of the pad. In some cases certainly it is not needed. In very long-standing cases we shall sometimes do no good with it. But in the majority of these young children who squint the pad is a most useful adjunct to the glasses.

Lastly, there is the question of operating. When ought we to operate? Some people still think that the treatment of squint consists merely of a little skilful cutting. They wish to bring the child on some convenient day to have an operation performed at once, and to take him away cured. One has to explain, of course, that the first step must be an examination; that probably glasses will do good; that the squinting eye will probably need to be educated for a while before any operation is undertaken. On the other hand, some surgeons say, "Never operate till five or six years of age." Others would make it ten years of age. These late operations sometimes give very good results, but this happens only where the functions of the squinting eye have been originally well developed and retained. Some cases which appear to be good cures are not really so; the eye is put straight, or nearly so, but its functions remain to a large extent in abeyance. In some at least of these latter if treatment, including operation, had been undertaken earlier, the result as regards the function of the eye would have been better. The rule should be to cure the squint without an operation, if possible; that is to say, to treat it by the persevering use of glasses, and by educating the squinting eye; to watch the progress systematically, and, so long as improvement goes on, to do nothing more; but when progress by these means comes definitely to a standstill, and an operation offers the only prospect of recovering binocular vision, then to operate whatever be the age of the child. Of course, we have to remember that an ill-judged or a too free division of the tendon may lead to divergence later on, but this should very seldom happen, and when it does happen it is not serious, because it is easy to readjust the tendon. It is far easier to bring a muscle back into its proper place than to develop a function which has been arrested in early life.

The methods of operating need not be discussed here. To those who do not practise as ophthalmic surgeons the question where to seize a tendon, how to cut, and so forth, is of no great interest.

Briefly to sum up the whole matter: the common strabismus of children begins very early in life, most commonly at three years of age, but often a little earlier or a little later. Many cases are neglected in the early stage. (Among a large number of cases tabulated by myself a few years ago, nearly half had been left without treatment for at least three years.) Delay of treatment, especially in the cases which begin very early, involves disturbance of development in the visual

Digitized by Google

apparatus which is often irremediable later on. Every squinting child, therefore, should be examined as soon as possible after the squint begins. In addition to the use of glasses, and to operation where this is required, we ought to ensure that the squinting eye has the benefit of compulsory education. If the two eyes cannot be educated together as they should be, they must be educated turn and turn about. In this way only can the maximum amount of benefit be obtained.

LEBESGUE reports five cases of fracture of the patella, which were treated with pads above and below the fragments, a long zinc rod behind the leg, and an extension apparatus, all held in place by bandages. These are kept in place five or six weeks, when the patient is cured.

VERNEUIL has used kelene, pure chloride of ethyl, in seventy cases to produce general anæsthesia. A special mask will be needed for its administration. Verneuil has thus far only used it in operating for a short time, changing to chloroform if the operation should be prolonged. The great advantage of chloride of ethyl is that it produces anæsthesia at once, while ether and chloroform waste much time before the patient is anæsthetised. It is even more rapid than bromide of ethyl. Nor do any disturbances follow its use. Verneuil forms no conclusions yet, as he intends making further experiments.

WILLEMS has collected fourteen cases from the literature, in which the omentum was sutured to the abdominal wall in order to deplete the portal circulation by establishing an anastomosis of the blood-vessels by which the ascites, in cirrhosis of the liver, will be relieved. In nine of these cases the effect upon the portal circulation was excellent, and recovery followed. Willems operated on a child of five years, with a large mesenteric tumour, without ascites. The abdominal wall was denuded of its peritoneum, and the omentum sutured to it. Both the spleen and liver decreased in size since. The child has been improving ever since the operation. Willems wonders whether this proceeding may not predispose to internal strangulation.

Philadelphia Med. Journ., August 23rd, 1901.

GONORRHŒA IN WOMEN.

A Clinical Lecture delivered at Charing Cross Hospital, June 20th, 1901,

By T. W. EDEN, M.D.,

Assistant Obstetric Physician to the Hospital.

GENTLEMEN,—It is due largely to the clinical researches of a German physician, Næggerath, who practised in New York some twenty-five years ago, that we know as much about gonorrhœa in the female sex as we now do. He astonished the medical world by publishing views as to the frequency of this disease in women, which were very different to what had been generally held up to that time. But his views served to direct attention to the subject, and further examination and research has shown that gonorrhœa is a very important condition in women, and may lead to extensive and permanent damage to the sexual organs. The results of this disease vary very much in different cases, and occasionally the diagnosis is a matter of considerable difficulty. We shall consider first the diagnosis, then the clinical varieties, and lastly the treatment.

In the majority of cases there is not very much difficulty about the diagnosis of this disease in women, and yet cases do occur in which the difficulty may be very considerable. Knowing as we do that there is a specific organism, the gonococcus, it appears at first sight that there ought not to be very much difficulty in diagnosis. But when we come to speak of the organism from the practical point of view, we shall see it is not of the same importance for diagnosis as many other organisms; for instance, as the tubercle bacillus is in the case of tuberculosis.

The points upon which we rely in regard to the early diagnosis of gonorrhœa are, first of all, the *characters of the discharge*, and secondly the *distribution of the inflammation*. In the early stage the discharge is almost pure pus, and the patient complains of a "yellow" discharge. I would emphasise the importance of the medical man always satisfying himself as to the character of discharges from the female genital tract. There is nothing so common as the complaint of a discharge, but often the account given by the patient herself as to the discharge from which she is suffering is misleading. It is necessary

Digitized by Google

that you should satisfy yourself as to the characters of the discharge. The number of conditions which give rise to a purulent discharge from the vagina is not very great; that is to say, there are not very many different conditions which you will have to differentiate from gonorrhœa. Whitish discharges, thick or thin, and slightly coloured discharges, are very common, but a discharge of pure pus from the vagina is not common. Another point about gonorrhœal discharge is that, although it is purulent, it is not fœtid. The majority of purulent discharges from the vagina are characterised by fœtor, and the fact of there being no odour will at once enable you to write off two or three conditions which give rise to purulent discharges which are always fœtid. For instance, a pelvic abscess which has ruptured into the upper part of the vagina, be it a pyosalpinx, or an ovarian abscess, or a suppurating ovarian cyst, will give rise to a purulent discharge, but it is almost certain to be stinking. Again, in septic conditions following abortion or labour, a purulent discharge is not uncommon, but there also the discharge is stinking. One of the commonest conditions which gives rise to a purulent discharge in women, apart from gonorrhœa, is a neglected vaginal pessary, and in that case also the discharge is offensive. When you think of the conditions which will give you a purulent discharge which is not fœtid, there are, practically, only two; one is gonorrhœa, and the other is simple acute vaginal catarrh. Now, an acute catarrhal inflammation of the vagina which is not gonorrhœal is uncommon, but still it does occur, and when it occurs, the greatest possible care has to be taken to distinguish it from this disease. It is distinguished partly by the characters of the discharge, but mainly by the clinical course of the disease. This brings us to the question of the organism, because the presence or absence of the gonococcus will often be the point upon which you will expect to rely to distinguish gonorrhœal vaginitis from simple catarrhal vaginitis. The organism is a diplococcus, and as seen under the microscope it consists of pairs of small oval-shaped bodies, with an outer convex and an inner flat surface, the flat surfaces being apposed. They are situated in the pus cells and in the epithelial cells which are found in the discharge. When this organism was discovered by Neisser, it was supposed to be pathognomonic of gonorrhœa. But further observation has shown that other diplococci occur which are indistinguishable except by culture experiments from the gonococcus. Indeed, an organism almost exactly like it has been cultivated from the healthy male urethra. The differential diagnosis of the diplococcus of gonorrhœa from other diplococci can only be accomplished by a skilled bacteriologist. It is quite possible for the clinical physician, when he sees a case which he believes to be gonorrhœa, to obtain a specimen of the discharge, stain it in the ordinary simple way, and recognise the presence of a diplococcus which sufficiently resembles the gonococcus. For medico-legal purposes it is not enough, however, because other organisms which are like diplococci in their staining reactions must be differentiated by culture experiments before one can be sure of the disease. The mere recognition of the organism in the discharge is therefore comparatively useless for diagnostic purposes. Some time ago I saw a girl of seventeen who alleged that she had been seduced, and in consequence was suffering from a disease. She had noticed a yellow discharge for two months, and she presented at first sight many of the ordinary clinical features of gonorrhœa. There was a subacute inflammation in the vulva and vagina, and there was a free purulent discharge. Afterwards with two colleagues I examined her under an anæsthetic, and then found that although there was obviously an acute vulvo-vaginitis with a free purulent discharge, there was no urethritis or urethral discharge, no inflammation or thickening of the Bartholin glands, or discharge from them on pressure. On passing a small speculum I found the secretion in the cervical canal was healthy, and the inflammation had not spread to it. On microscopical examination of the discharge no diplococci could be found in it. So on further examination the case for gonorrhœa was, from a medico-legal point of view, quashed. I have little doubt in my own mind, from a clinical point of view, that the disease was gonorrhœa, and after the lapse of two or three months the diplococcus is usually absent from the discharge. But, if you are put into the witness box, and asked to state whether a disease is gonorrhœa or

Digitized by Google

not, the wisest plan, unless you have complete bacteriological proof, is to say that you cannot swear, and that it is impossible with the means at your disposal to swear that it is either the one thing or the other.

Passing now from the nature of the discharge, the next point concerns the distribution of the inflammation. A curious thing about gonorrhœa in women is that the stress of the inflammation is very apt to fall on different parts of the genital tract. In man the disease runs a uniform course; the whole urethra is involved, and if the disease lingers, it is in the membranous or prostatic portion. But in the case of women it is very different. The commonest variety of the disease is that in which the principal symptoms arise from inflammation of the vulva. This is the variety which, being the commonest, we shall see most frequently. It is stated by writers on the subject that cases occur in which the vulva and the vagina absolutely escape, and are not involved in the inflammation at all, the primary inflammatory lesion being in the cervix. Now, although I have seen cases in which the uterus has been very severely involved in gonorrhœa, I have never seen one in which the primary lesion was uterine, and there was reason to believe that the vulva and the vagina escaped. In other cases, curiously, the stress of the disease falls not upon the vulva, but upon the uterus or ovaries, the tubes, or on the peritoneum. I have seen a case—which I will tell you more fully about soon—of acute general peritonitis resulting from gonorrhœal inflammation of the tubes, which was mistaken for a perforated vermiform appendix, and which was operated upon under that impression.

The next point to remember in regard to the distribution of the lesions is that gonorrhœal inflammation shows a remarkable tendency to spread through every possible channel that is open to it. Of course gonorrhœa arises from the implantation in the genital canal of gonorrhœal discharge. Usually that gonorrhœal discharge is deposited in the vulva or the vaginal canal, and usually the inflammation first of all appears in the vulva and in the vagina. When you consider the anatomical relations of the vulva, you will see there are a number of channels through which the disease may spread from this part. There are a pair of fair-sized glands lying at the sides of the vaginal outlet—Bartholin's glands—with ducts which open outside the hymen. These glands are, in the majority of cases, involved, to a greater or less extent, in gonorrhœa. Then there is the patent urethral canal—a straight canal leading to the bladder, and that is almost invariably affected in gonorrhœa. The affection, both in the case of Bartholin's glands and the urethra, is by continuity; the inflammation creeps through the patent meatus and down the Bartholin ducts, and so affects these glands and the urethra.

In the early stages, while there is free purulent discharge, acute suppuration of Bartholin's glands may occur, forming a labial abscess. Usually the glands are not involved until a later period, and even then do not become acutely inflamed. It is rare, however, for them to escape entirely, and careful examination will reveal certain changes. On pinching up the labium you will probably feel that the gland is thickened and enlarged, and on squeezing it you will find you can squeeze out a certain amount of discharge from the duct which may have the same characters as the vaginal discharge, or it may be more distinctly purulent. But a Bartholin abscess may form even weeks or months after the time at which the disease was contracted. The inflammation spreads very early to the urethra, and is apt to linger there when it is resolved in the vagina and the vulva. The inguinal chain of glands may become infected by lymphatic absorption from the vulva, causing a bubo. From the vagina, of course, it spreads easily by continuity through the external os to the cervical canal, and in untreated or imperfectly treated cases this usually occurs. Cervical gonorrhœa is a lesion which is not often spoken of, but is a very definite and well-recognised lesion which you will frequently find. Sometimes the inflammation in the vulva and vagina will subside fairly rapidly, but considerable purulent discharge continues. At first sight it is not very clear where the discharge comes from, but if you pass a speculum and expose the cervix, you will find that there is a free discharge of pus coming from the cervical canal. It usually has the tenacious characters of the ordinary cervical secretion, but instead of being clear and transparent it is obviously pus. Under the microscope it looks like pure pus, and you may find in it a diplococcus like that of gonorrhœa. From the cervix

Digitized by Google

the gonorrhœal discharge is likely to go on for a long time after the disease has cleared up in the vagina, and it is a lesion which it is impossible for the practitioner to cure by merely douching the vagina. From the cervix the disease may spread to the body of the uterus. Uterine gonorrhœa, as distinguished from cervical gonorrhœa, I believe to be one of the rarest forms, but I saw a very striking case of this kind a year or two ago. The patient was a young married lady, and five months after her first confinement she began to suffer from a yellow discharge, with a great deal of pain. At the time this happened her husband was suffering from a similar discharge from the urethra. He absolutely denied any impure sexual intercourse, and he stated that there was a little doubt as to whether the discharge appeared first in his wife or in himself. I did not see her until the discharge had continued for nearly a month; about a fortnight after it began a swelling formed in the vulva and burst, discharging a considerable amount of pus and blood. It may have been a Bartholinian abscess, or some other form of labial abscess caused by the inflammation; probably the former. When I saw her there was not much inflammation of the vulva and not much in the vagina. The mucous membrane was pale and fairly healthy, and there was no tenderness. There was a good deal of discharge on compressing the urethra, and there was also some thickening and tenderness of the Bartholin glands. On passing a speculum an enormous quantity of fairly thick purulent discharge was found lying in the vaginal fornices; three or four tablespoonfuls were mopped out. On clearing away the discharge which lay there, pus could be seen pouring out through the external os. The uterus was big and extremely tender, and the discharge was not nearly so thick as you find it in a simple case of cervical gonorrhœa. On account of the size and tenderness of the body of the uterus, and the amount of pus which was coming away, and the thinness of the pus, I thought it was coming more from the body of the uterus than from the cervix. It was uterine gonorrhœa, and from the amount of discharge which was coming away many ounces of pus must have been discharged from the uterus daily. I arranged to examine her more carefully, a day or two afterwards, under an anæsthetic, but I was not able to do so, because on the day after I saw her, her temperature suddenly went up to 105°, and she had an acute illness for two or three days, with severe abdominal pain and distension. I did not see her during this illness, but I have no doubt it was an attack of pelvic peritonitis. I saw her when the attack was over, and then I examined the condition of the appendages, and found the uterus retroverted and partly fixed, and both tubes and ovaries matted, and lying in the pouch of Douglas. This was little more than a month after the onset of the disease, and in that time it had travelled from the vulva, where it attacked the urethra, to the Bartholinian glands, from the vagina to the uterus, thence to the tubes and ovaries, and the peritoneum. In this case the stress of the inflammation at the time I saw her was in the uterus. Occasionally the stress of the disease falls even higher up, on the tubes and ovaries. I saw a striking case of this kind about two and a half years ago in a girl aged eighteen, who for some months had been suffering from a purulent discharge. Very little notice was taken of this, and I think very little treatment was adopted. She was a servant, and was suddenly seized with severe abdominal pain while doing her work. The doctor in attendance diagnosed appendicitis, and she became so ill—signs of acute general peritonitis supervening—that it was thought the appendix must be perforated, giving rise to a general suppurative peritonitis. She was sent into the hospital, where the abdomen was opened, in the belief that it was a case of perforated appendix. The abdomen was opened in the right iliac fossa, and when the appendix was reached it was found to be free from adhesions, and although acutely inflamed like the rest of the peritoneum, it was otherwise intact. The operator then explored the pelvic organs, and on pulling up the tube it was found to be acutely inflamed, and pus was dripping from the abdominal ostium. The right tube and ovary were removed, and with considerable difficulty the other tube and ovary were brought out of the same opening, found to be in the same condition, and removed. There was no occlusion of the abdominal ostia, and therefore the pus, instead of being confined in the tube and forming a pyosalpinx, flowed into the peritoneal cavity and set up general acute peritonitis. I do not suggest that cases like this are very common, but they do occur occasionally, and it is rather a striking illustration

Digitized by Google

of the fact that the stress of the gonorrhœa may fall in different places; although in the commonest cases it is in the vulva or vagina, it may be in the uterus, and it may be in the tubes and pelvic peritoneum.

The rate at which the inflammation spreads is a point upon which I have not very much information. The case I have related to you of uterine gonorrhœa is the clearest instance which throws any light upon it that I know of, and in that the inflammation had reached the pelvic peritoneum within a month of its onset. The length of time which elapsed in the case of acute general peritonitis leads me to believe that the infection reached the peritoneum in that case through the lymphatics. In the case of uterine gonorrhœa, of course, the disease reached the uterus by direct spread from the vagina, but as there was no purulent affection of the tubes and ovaries, only an adhesive peritonitis, I think the tubes and peritoneum had become involved by lymphatic absorption from the uterus, and not by direct continuity, or we should have found a suppurative condition of the tubes and ovaries. Gonorrhœa spreads, then, either by direct continuity or by lymphatic absorption, and the spread by direct continuity is much more rapid and acute than by any other method of spread.

The *symptoms* to which gonorrhœa gives rise I have purposely left until now, because I do not think they are of nearly as much importance as the physical signs. The more one sees of the diseases of women the more one becomes convinced that much less attention should be paid to symptoms than to the physical signs. In the case of gonorrhœa the symptoms to which it gives rise are sometimes almost insignificant. This is usually the case when the vulva and vagina are not very acutely inflamed. The amount of discharge which the patient has is inconsiderable, and she is not very much inconvenienced by the disease, except that there is usually scalding pain on micturition. Of course if the vulva is acutely inflamed the symptoms are much more severe. When the uterus and appendages become involved in spreading gonorrhœa, we find there is profuse and extremely painful menstruation. The onset of such symptoms in a case in which you have reason to believe there is a history of gonorrhœa is an indication for very carefully exploring the condition of the uterus and the uterine appendages. Pelvic peritonitis is a complication which, of course, gives rise to symptoms which are so marked that it is impossible to overlook them. General peritonitis is rare as a result of gonorrhœa, but attacks of localised pelvic peritonitis are fairly common. The symptoms to which they give rise are similar to those of general peritonitis, but less marked. The amount of distension is less, and the amount of general disturbance that the patient suffers from is very much less too.

Now, when we come to consider the question of the *treatment* of this disease, one has to admit at the onset that the treatment is not very satisfactory. This applies, I suppose, to a certain extent to the treatment of the disease in men also; but in women the difficulty is even greater. In the first place, drugs are absolutely unreliable. I suppose drugs of a certain class are regarded as very useful in the treatment of the disease in men, drugs which are excreted through the urine and are capable of being thus brought into contact with the inflamed mucous membrane; but gonorrhœal urethritis in the female, instead of being the essential lesion of the disease, is only an incident in it, and a comparatively unimportant one; therefore not much benefit is to be expected from such methods.

There are no other drugs which can be given by the mouth that are of very much benefit. Treatment, to be serviceable, must be local treatment. The commonest device by which we try to cure gonorrhœa is the vaginal douche. But douching is entirely inadequate to cure this disease, and the way in which most women use the vaginal douche renders it almost valueless. They like to use it in the stooping position, because it is least trouble; the tube is passed half-way up the vagina and the lower part of the canal is washed out with the solution. If a vaginal douche is to be taken and the full benefit obtained, it must be taken when the patient is lying on her back. It is only by using it thoroughly in this way that very much benefit can be derived from it. But even used in this way douching is very inadequate as a means of curing vaginitis. The vagina, as you know, is not a smooth-walled canal; it is thrown into ridges and folds, and the mere passage of water over it does not cleanse it. It is necessary to do more than that in order to remove the discharge from the mucous surfaces, and the method

Digitized by Google

which gives the best results is disinfection of the vulva and vagina under an anæsthetic. It is too painful to be done without an anæsthetic, so ether or chloroform must be given, and the disinfection has to be carried out very thoroughly. You try to disinfect—using the term in its surgical sense—the whole vulvo-vaginal canal from the cervix to the vulva. A duck-bill speculum must be used, and a strong antiseptic solution must be thoroughly rubbed into the vaginal walls. A very useful implement for this purpose is a tooth-brush, and with this the vaginal walls can be thoroughly scrubbed, and the antiseptic brought into contact with all the ridges and folds of the mucous membrane. The best antiseptic is a strong solution of perchloride or binodide of mercury, about one in 1000, which should be rubbed thoroughly into the vaginal walls until they begin to ooze. The best chance of success is to do this in the early stages before the Bartholinian glands or the cervix are involved. If the Bartholinian glands are involved you cannot treat them in the same way, and you leave behind a focus of infection. And if the urethra is involved you cannot treat that in the same way, but the urethra can be dilated gently, and after that it can be swabbed out with a milder antiseptic, such as one in 2000 of perchloride. I have done this in many instances, and always with good results, supposing that the disease is confined to the vulva and vagina.

If patients decline this method, then I think the best kind of treatment is by means of medicated vaginal pessaries. A pessary made of gelatine or ordinary cocoa butter, and containing twenty grains of iodoform and ten grains of oil of eucalyptus, forms a good application. This can be passed into the vagina when the patient goes to bed; it melts, and the solution flows over the vaginal walls and into the folds and depressions. Douches can of course be used in addition.

In cervical and uterine gonorrhœa the treatment is very much more difficult than it is in the case of vaginitis. It is impossible to disinfect the uterine cavity as thoroughly as the vagina. The best treatment I believe is somewhat on the same lines as the treatment of the vagina. In the case of uterine gonorrhœa which I related to you the treatment we adopted was to partially dilate the cervix sufficient to allow a double-way tube to be passed, through which the uterus could be irrigated. We thoroughly irrigated that uterus twice a day with a fairly strong antiseptic solution, and after washing away all the purulent discharge from the uterine cavity, an application of strong iodine was made to the endometrium. I have tried curetting frequently in similar cases with good results; but I do not think curetting gives as good results as irrigation with strong applications to the endometrium.

With regard to internal complications, such as peritonitis, and disease of the tubes and ovaries, these cases can only be treated on general principles. Surgical interference is required if you have reason to believe that pus has formed in the tubes and ovaries, but palliative treatment can be tried if you believe pus is not present. Opinions differ as to the extent to which cases of diseased tubes and ovaries should be subjected to operation. I have tried both plans fairly extensively, and I am more in favour of removing the tubes and ovaries in such conditions than of leaving them; but one often has to be guided by the circumstances of the patient. Of course a working woman, who has to earn her living, cannot afford to be invalided, and in her case operation, affording as it does a means of getting rid of her trouble once for all, is desirable and justifiable, whereas in women who are better placed in life you may not feel justified in advising them to take the risks of a radical operation.

Picric Acid in Smallpox. J. F. Romero.—In 162 cases of smallpox, 46 recovered without noticeable scars, and 101 without a single trace of pitting. All had been treated with picric acid applied as a lotion or in a salve, and pitting occurred in only 15 cases. The picric acid probably acts by destroying the pyogenic germs that may find their way into the pustules. In a number of cases the confluent pustules covered the exposed parts of the body and the legs, but under the picric acid they subsided without a trace. In many instances he noted the coincidence that other members of the family failed to contract the smallpox from patients duly treated with picric acid. His formula for the lotion is 2 gm. of picric acid in 15 gm. of alcohol and 185 gm. of water.—*Cronica Medica Mexiana* (Mexico), August 1st.

Digitized by Google

TWO CLINICAL LECTURES
ON THE
CONTROL OF ARTERIAL HÆMORRHAGE AND THE TREATMENT OF ANEURYSM.

Delivered at the Post-Graduate College,
West London Hospital,
By W. McADAM ECCLES, M.S., F.R.C.S.,
Senior Assistant Surgeon to the Hospital; Assistant Surgeon, City of London Truss Society, etc.

LECTURE I.

GENTLEMEN,—I am announced to lecture to you upon the control of arterial hæmorrhage and the treatment of aneurysm. And to-day I propose to deal solely with the first part. By the wound of an artery loss of blood occurs. This may be either subcutaneous, when a vessel is either torn or cut beneath the skin, or external, when an artery is divided through an open wound. When an artery is injured, it may be either completely or only partially divided. The difference in the amount of hæmorrhage from a complete transverse division and an incomplete is very marked. In a complete division there is a condition of the vessel which tends considerably to the arrest of hæmorrhage because the divided ends can contract and retract, the contraction being due to the circular muscular fibres of the arterial wall, the retraction within the sheath of the artery being caused partly by the activity of the longitudinal muscular fibres and partly also by the natural elasticity of the vessel. In the incomplete transverse division, however, this natural elasticity and the action of the longitudinal fibres will tend to cause the wound in the vessel to gape, and therefore hæmorrhage to continue for a longer time than it would in the case of complete transverse division. On the other hand, supposing a vessel is incised longitudinally the circular muscular fibres of the vessel wall do tend to cause a widening of the wound, but not to such a degree as do the longitudinal muscular fibres in the incomplete transverse division; so that hæmorrhage from an artery wounded longitudinally is not as a rule so severe as hæmorrhage from an artery incompletely transversely divided. In order to understand the arrest of arterial hæmorrhage from a surgical point of view, one must have some clear idea as to how Nature controls this form of bleeding. All hæmorrhage may be arrested in one of two ways, either temporarily or permanently. Nature proceeds first of all to the temporary arrest, and then to the permanent arrest, and I venture to think that the surgeon can only help Nature in the temporary arrest of hæmorrhage. What then are Nature's methods for the arrest of hæmorrhage? First let us begin with temporary arrest. The methods employed by Nature in the temporary arrest of arterial hæmorrhage may be divided into two, the local and the general. The local Natural means of arrest of arterial hæmorrhage consists of four distinct factors. Two of them I have already mentioned, viz. the contraction of the artery so that its lumen becomes considerably diminished in size, and the retraction of the vessel within its sheath so as to leave a very rough surface beyond. Thirdly, there is the incurling of the internal and part of the middle coat. This incurling naturally still further diminishes the opening from the divided vessel, and also tends to make another rough surface, over which the escaping blood must flow. Then follows the fourth point, the formation of an external clot. The blood, as it pours out from the divided vessel, meets with the roughened walls of the inner surface of the sheath, and of the tissues outside. And when blood comes in contact with tissues other than the smooth living internal wall of an artery, it tends to deposit fibrin, to become coagulated, leading in this case to the formation of an external clot. As the external clot gathers, so the channel through which the blood passes becomes smaller and smaller, till at last the passage outside the vessel in the sheath becomes occluded with coagulum. Subsequent to the formation of the external clot, an internal clot is deposited within the vessel lumen, and usually extends as high up as the first fair-sized collateral branch. The clot will often pass beyond some of the smaller collateral branches, but in certain cases where an artery has no branch for some considerable distance on the proximal side, the clot may stop short of a collateral branch; but, as a rule, it reaches up as far as the first fair-sized collateral branch, and tends to assume a conical form. The external and internal clot may be likened to the two parts of a cork, while the artery itself represents the neck of the bottle, the external clot representing the portion of cork that lies above the neck, and the internal clot the portion that lies actually

Digitized by Google

within the neck. And any cause whereby the clot (external and internal) is displaced, leads to the second form of arterial hæmorrhage, known as reactionary hæmorrhage. These are Nature's local methods in the temporary arrest of arterial hæmorrhage.

The general methods that Nature adopts to arrest hæmorrhage are three. First, the diminution in the force and frequency of the beat of the heart. Secondly, syncope; the patient becomes faint from loss of blood and diminution in the flow to the central nervous system, and tends to fall into a horizontal position; in other words, the patient faints and falls, and this leads to rest. The patient is now no longer able to move about, with the result that there is less likelihood of disturbance of the clot. Lastly, the more blood that is lost, up to about half the quantity of the blood in the body, tends to induce a condition of the blood which renders it more easily coagulable. After securing temporary control, Nature proceeds to the permanent arrest of hæmorrhage, and to put it very briefly, there are two stages in the permanent arrest, first, the formation of interstitial granulation tissue, and, secondly, the conversion of this into fibrous tissue. The external and internal clot practically act as if they were foreign bodies. Interstitial granulation tissue is produced, and provided there is no septic interference the interstitial tissue very quickly becomes converted into fibrous tissue. The coagulated non-circulating blood is therefore replaced by living fibrous tissue, the blood-clot being to all intents dead matter, and merely acting, as it were, as a foreign body. It is not infrequently said that the blood-clot forms the fibrous tissue; this is not true; it is the cells which permeate the blood-clot which give origin to the fibrous tissue. It may be graphically said that the coagulum is only the scaffolding, and that the proliferated cells are really the material with which the house is built, and that the scaffolding disappears as the house progresses. This is the permanent arrest of hæmorrhage, and unless it is brought about, unless the vessel is occluded by a fibrous tissue plug adherent to the vessel wall, there can be no permanent arrest of arterial hæmorrhage, and it is for this reason that I have ventured to state that the surgeon is altogether unable to permanently arrest hæmorrhage.

After this very brief review of Nature's method of arterial hæmorrhage arrest, I wish to discuss the surgeon's methods of temporary arrest. There are three chief ways whereby the surgeon can temporarily control hæmorrhage so as to allow Nature time to bring about a permanent arrest. They are: pressure, ligature, and torsion. Pressure may be applied first of all directly on the injured vessel, through the wound, and this is one of the most usual methods and one of the most satisfactory methods of temporary arrest of hæmorrhage. Secondly, pressure may be applied on the main artery on the proximal side of the wound, that is the heart side of the injury. There are several varieties of ways in which pressure may be applied directly to the vessel. Digital pressure is one. A wound, say in the middle of the forearm, is discovered, and arterial blood is issuing forth. Naturally, the first thing to do is to put a finger over the actual bleeding spot and apply pressure in that way; curiously enough, this method is frequently forgotten as the first means of arrest of hæmorrhage. Then, supposing there are instruments at hand, the next plan, and the best method of arresting hæmorrhage in a wound, is to seize the bleeding vessel with a pair of pressure-forceps—Spencer Wells' forceps,—which hang on to the vessel spontaneously when once attached. These are the two chief methods of local pressure. Of course, I am speaking purely of arterial hæmorrhage, but other forms of pressure may be employed with regard to veins and capillaries.

Pressure may also be applied on the main vessel on the proximal side, and for a wound of an artery in the forearm it would be needful to apply pressure on the brachial in the arm, or on the subclavian in the neck.

The second method of the surgeon for temporary arrest is to ligature. The materials which are used for the ligature of arteries are various. Silk, in the form of Chinese twist, is certainly the most usual and convenient substance. Gauges Nos. 0 and 2, are in common use, the first does for all vessels practically up to the size of the lingual, while size No. 2 does for larger vessels, saving the largest. Some of the advantages of silk as ligature material are these:—First of all, it is easily obtainable. Secondly, it is very readily tied. Thirdly, for its thickness it is very strong. Fourthly, it can be rendered absolutely aseptic by boiling. Fifthly, it can be very easily kept without very much danger

Digitized by Google

of its becoming disorganised in antiseptic solution. On the other hand, it has the disadvantages of not becoming, as a rule, absorbed, it becomes encysted rather than absorbed. Possibly, also, it may by remaining become infected at a later period, and then become extruded from the wound. Another material that is used for ligature is catgut, prepared so as to render it aseptic, chiefly by what is known as the formalin method. Sulpho-chromic catgut, sometimes called green catgut, is also used. One of the disadvantages of catgut is that it is very difficult to render aseptic, and that it is liable to be uncertain in the length of time that it will remain in the tissues before it becomes absorbed. Other materials are used, such as kangaroo-tail tendon, ox aorta, and more recently gold-beater's skin, drawn into a thread. However, these are more commonly used for the largest arteries.

Thirdly, there is torsion. This may be used in two ways: the first is called free torsion, where a small artery is seized by a pair of pressure-forceps at its end, and the forceps are twisted round until they come off. This method of free torsion is very useful indeed for quite small vessels. The other method, which is known as limited torsion, is that used for large vessels, such as the femoral artery. It would not do in these cases merely to seize the vessel at its end and rotate the forceps; but it is necessary to seize the vessel, first of all, transversely to its long axis by a pair of forceps, secondly, to seize its open end with another pair of pressure-forceps, and then twist the small piece of artery between the two forceps, turning the forceps for at least six times, but not twisting the end of the vessel off.

As I have indicated, the varieties of arterial hæmorrhage are generally given as three. They are primary, reactionary, sometimes also called recurrent, sometimes intermediate, and lastly, secondary hæmorrhage. It is well to have a clear idea as to what is meant by these three terms. Primary arterial hæmorrhage is the loss of blood immediately following upon the wound of an artery, and may be either subcutaneous or open. Reactionary hæmorrhage, I think, is best defined as the hæmorrhage which occurs owing to the failure of Nature's or the surgeon's methods of temporary arrest. The general definition of reactionary hæmorrhage is the hæmorrhage which occurs during the first twenty-four hours after an injury to an artery, but this is not very scientific. It is also defined as the hæmorrhage which occurs during the period of reaction; then it is necessary to define what is meant by the period of reaction. Secondary hæmorrhage, I think, is best defined as hæmorrhage which occurs owing to the failure of Nature's methods of permanent arrest. That is to say, anything which causes failure in the formation of the adherent fibrous tissue plug produces secondary hæmorrhage. It is also defined as the hæmorrhage which occurs after a period of reaction, or the hæmorrhage which occurs after twenty-four hours from the wound of a vessel, neither of which are very satisfactory definitions.

I propose in the remainder of the time at our disposal to deal with some points in the arrest of hæmorrhage from special arteries. What has been said was intended to lead up to this special discussion. Obviously the time is not sufficient to speak of every artery in the body, but the treatment of arterial hæmorrhage from the palm of the hand will be first reviewed. It is well to remember that in the palm of the hand there are two arterial arches, the superficial and the deep palmar arch. The superficial palmar arch is formed by the superficial volar branch of the radial, joined by the superficial branch of the ulnar. The lowest limit of this arch is indicated by a line drawn across the palm at the level of the outstretched thumb. This arch gives off the digital branches. The arch lies beneath the deep fascia, the palmar fascia, but superficial to all the other structures in the palm of the hand. The deep palmar arch, on the other hand, lies beneath all the flexor tendons and the median nerve, and is practically one of the deepest structures of the palm. It is formed by the radial, which having turned round the outer side of the wrist, passes through to the front to reach the palm, between the two heads of the first dorsal interosseous, the termination of the radial being joined by the deep branch of the ulnar, and the position of this arch is one finger's breadth nearer the wrist, that is to say higher up, than the superficial arch. From the deep arch there are given off numerous branches, particularly the interosseous ones running forwards, and the recurrent branches which run upwards to join the anterior carpal arch, formed by the anterior carpal arteries of the ulnar and of the radial. This anterior carpal arch is also joined by the termination of the anterior interosseous artery.

Digitized by Google

so that there is an important cruciform anastomosis. In a punctured wound of the palm of the hand there may be involved either the superficial or the deep arch, or one of their branches. The hæmorrhage from a punctured wound of the superficial arch or its branches is a matter that is usually very easily dealt with and controlled. All that is necessary is to cut down upon the vessel and ligature the artery on both sides of the wound in its wall. On the other hand, in a punctured wound of the palm which causes an opening into the deep palmar arch, the vessel lies so deeply that it is not so easy a matter to expose and tie the vessel, but certainly in these days of asepsis this should, as a rule, be attempted. First of all, put an Esmarch's rubber bandage from the hand to the middle of the arm, so as to exsanguinate the limb, and here an elastic tourniquet. The palm of the hand being now bloodless, a careful dissection is to be made, so as to avoid damaging tendons or nerves, down to the site of the deep arch. With retractors the tissues are held well apart, and then, by slightly loosening the tourniquet, the wound in the vessel may be recognised by the blood spurting out of it. With an aneurysm needle ligatures may be drawn underneath it, and tied on both sides of the wound. Supposing there are not facilities for dealing with the wound in this way, then it may be necessary to treat it by local pressure, and local pressure, if properly carried out, will in the majority of cases arrest the hæmorrhage; although it is possible it may not do so, and it may cause even more damage to the tissues in the palm of the hand than cutting down and tying the artery. Immediate control is obtained by pressure upon the brachial artery in the arm, so that the patient loses no more blood. Whilst this is being held, prepare the materials with which the pressure is to be applied to the palm. They are a conical pad of antiseptic gauze, some finger and broader bandages, a broad splint—that is one sufficiently wide to take the palm of the hand and to extend a little over it (about four inches in breadth),—and a sling. Proceed to wash out the wound with an antiseptic solution, and to cleanse around the wound, so as to get the parts as aseptic as possible. Then each finger and the thumb should be bandaged separately; otherwise there is great liability of œdema of the fingers. Then, placing the antiseptic dressing or conical pad into or over the wound, as the case may be, adjust the broad splint on the dorsal aspect of the hand, and bandage the conical pad and hand together against the dorsal splint. In this way pressure upon the wound is applied directly from before backwards. If no splint is used there would be a tendency for the hand to become concave, and so cause the conical pad to be lifted from the wound. Having applied the conical pad and bandaged the hand to the splint, flex the forearm so as to apply a certain amount of pressure on the brachial artery at the bend of the elbow, and also to raise the hand so as to limit the force of the heart's beat in the arteries. Place the hand towards the opposite shoulder and put the palm and forearm into a sling. Pressure obtained in this manner is sufficient, as a rule, to arrest the flow of blood from the deep arch. But it is not sufficient to absolutely arrest the circulation in the hand. The pressure is left on for twelve hours, and then it is expedient to take off the splint but to leave the conical pad *in situ*: in other words, take off the pressure but leave the dressing undisturbed. Again flex the forearm and put the hand across the chest. If hæmorrhage should recur, or if the pressure should have failed to stop the hæmorrhage, then the only way to arrest the bleeding is to tie the brachial artery in the middle of the arm, or at the bend of the elbow. This, it will be seen, is an obvious necessity if the anatomy of the anastomosis at the wrist is remembered. Blood can be brought by the anterior interosseous, across the anterior carpal arch, through the recurrent branches to the deep palmar arch, and so hæmorrhage would tend to recur, and it is therefore necessary to tie the brachial in such a case.

Technique of Subcutaneous Injections of Iron. A. PLESSI.—The contradictory experiences of various clinicians with the subcutaneous injection of preparations of iron is probably due to the choice of the point where the injection is made. The citrate of iron and ammonium is preferred at Galvagni's clinic, where the results of this method of administering iron have always been brilliantly satisfactory. A series of 359 injections are tabulated, showing that only three out of fifty-four injections in the back were free from some after-disturbance, while nothing of the kind was observed with 288 out of 305 gluteal injections.—*Journ. A. M. A.*, August 24th, 1901.

Digitized by Google

Bone Grafting.—After the skin has been thoroughly purified, a free incision is made over the deficient bone. The thickened remains of the periosteum are incised, and a bed is made for the new bone. The wound is then stuffed with sponges to arrest the bleeding, and covered with gauze dipped in a $\frac{1}{2000}$ sublimate solution. It is better not to use a tourniquet, because it gives rise to prolonged after-bleeding, which is apt to separate the bone-grafts from the tissues; the oozing should be allowed to stop before the grafts are inserted. The animal selected is then killed by an assistant and rapidly skinned, the carcase douched with $\frac{1}{2000}$ sublimate solution, and placed on an aseptic board or tray covered with a carbolised towel. With a fresh sterilised knife and forceps the muscles are rapidly peeled off the bone—the best one for the purpose is the humerus,—the periosteum being left undisturbed, and a portion is removed by cutting pliers. The length of the individual grafts is not a matter of much importance; they can be as long as the wound, but they must not be too thick or too broad, and it is well, therefore, to split the bone longitudinally into fragments with a stout knife. A series of these fragments are laid in the wound after removal of the sponges, until a sufficient amount has been introduced. The skin wound is then closed without a drainage-tube, an antiseptic dressing applied, and the part put on a splint, on which it is kept at rest sufficiently long for consolidation to occur. If the operation be done aseptically, no infection of the part will take place, and the pieces of bone do not separate; they gradually become welded together and united with the surrounding tissues, and to some extent they lead to the formation of new bone.

The above is taken from Part III of the 'Manual of Surgical Treatment' by Mr. Watson Cheyne and Mr. Burghard. The authors very truly say in the preface to this volume that in a work of this description for the practitioner the great essential is full and detailed information as to the best methods of treatment. Nothing can, of course, replace actual experience, but it is often of the greatest advantage to have a detailed record of that of others upon which to base one's work. It is this want that the authors have supplied, and Longmans, who are publishing the six parts in which the work is to be contained, well sustain their reputation for clear print and sound binding. Vol. III is in two divisions dealing respectively with surgical affections of bone and amputations.

Sajous's Annual and Analytical Cyclopædia of Practical Medicine.—Volume VI of this most remarkable production has now been issued by the F. A. Davis Company, Publishers, of Philadelphia, New York, and Chicago. The completing volume is quite up to the high standard of excellence reached by the preceding five, and Dr. Sajous is to be most heartily congratulated on the termination of his labours, which have worthily earned for him the admiration of all interested in the advancement of medical knowledge. We have so often drawn attention to the unique character of this annual, and have so often praised its utility to the practitioner, that we content ourselves this time with making a short extract from its pages on 'The Symptoms of Cancer of the Rectum.' The early symptoms of cancer of the rectum are far from being characteristic. Indeed, it often exists for a considerable period before the patient is cognizant of any trouble. Frequently patients consult a surgeon about hæmorrhoids or some minor affection of the rectum or anus, and it is only after a digital examination that the presence of cancer is recognised. Generally, the first sign experienced, in cases of this disease, is a sensation of uneasiness in the lower part of the back and along the inner sides of the thighs, with possibly a similar feeling within the rectum. At the end of a day's work this may be supplanted by actual pain of a dull, heavy character. The next and most usual symptom is morning diarrhœa. As soon as the patient gets out of bed, or soon thereafter, he is obliged to go to the closet. He may or may not pass any fæcal matter, but does pass a thin sanious discharge, having a most characteristic odour. Later the bowels move several times during the morning, and most of the stools consist of this sanious discharge. The colour of the stools is such that most patients ascribe their trouble to piles, and the discharge is attributed to blood from the latter. Constipation may alternate with the attacks of diarrhœa. Pain is nearly always a very late symptom of cancer of the bowel; this depends, however, upon the rapidity of the growth of the neoplasm. As soon as the growth invades the anal portion or neighbouring organs, or involves the sacral nerves, pain becomes a marked factor. Obstruction of the bowel and the straining efforts at defecation will tend to increase the pain. The discharge varies in quantity in different cases and at various stages of the development of the growth.

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 465. WEDNESDAY, SEPTEMBER 25, 1901. Vol. XVIII. No. 23.

CONTENTS.

PAGE

*A Clinical Lecture. By P. J. HENSLEY, M.A., M.D., F.R.C.P. 353

*Mechanical or Traumatic Arthritis. By W. ARBUTHNOT LANE, M.S.... ... 358

*Two Clinical Lectures on the Control of Arterial Hæmorrhage and the Treatment of Aneurysm. By W. MCADAM ECCLES, M.S., F.R.C.S. LECTURE II 363

Treatment of Carbuncle 368

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

Delivered at St. Bartholomew's Hospital, May 31st, 1901,

By P. J. HENSLEY, M.A., M.D., F.R.C.P.

GENTLEMEN,—I wish to call your attention to-day to one or two cases which I have under my care at present. One is a case in this hospital which I have headed pulmonary catarrh. I have done that rather because the immediate cause of the patient coming into Hospital was that condition. But the second heading, and the more important one, for the reason I wish to mention presently, is old pleural change with collapse of the base of the left lung. The patient is a little girl aged three years, and she was admitted on the 16th May, suffering from cough. On admission she was normal in aspect, was well nourished, and had a good deal of cough; the cough was not very frequent, but it was in troublesome fits. She was of a nervous temperament; her respiration was 44, but there was no distress. Forty-four, therefore, does not represent any respiratory trouble. There was no cyanosis. The tongue was clean, temperature but little raised, that is to say 99°. Pulse 100. There was found to be an impaired note over the left side of the chest, that is to say, more or less over the whole left side, the dulness becoming more marked lower down towards the axilla, and behind also there was well-marked basic dulness. Breathing was weak throughout, faintly bronchial in character, and on the whole weakest towards the base, with irregular mixed râles, somewhat of a creaking character in places. There was no definite friction; the side was somewhat fallen in; the movements were less on that side than the other. The percussion was good on the right side, and I do not know that one could note anything abnormal. It was a very much better percussion note than on the other side. The breathing was rather harsh in character, but not bronchial. There were some rather loose râles. The heart was displaced a little to the left, the apex-beat being somewhat outside the nipple line in the fourth interspace. This child had been under the care of Dr. Morley Fletcher, who had

Digitized by Google

seen her some months before, and from his notes I am able to fill up the history. The child appears to have had measles in January last year, and in June it was reported to have had a good deal of cough, spasmodic in character, and, what is unusual in a child so young, a thick yellow sputum freely expectorated. I call your attention to that, because it opens the question as to the origin of things. Dr. Fletcher appears to have seen her in October, 1900, and she was then well nourished, the fingers were not clubbed, there was no cyanosis, and the physical signs were as follows: left side front and axillary region and slightly behind more or less dulness, the dulness being most marked towards the base, with very faint breathing in front, somewhat faintly bronchial in character, especially near the left nipple. A certain scanty amount of crepitant sound at the upper part, near the apex. Posteriorly the breathing was weak, but there appeared to be no râles. The apex-beat at that time was outside the nipple line, somewhat in the position it at present occupies. The heart sounds were clear. Some of the sputum was obtained shortly after his seeing the child first, and it was examined, but no tubercle bacilli were found. In December last year he found that the dulness of the left base remained, and there was absence of breathing sounds; but there was not so much râle as there had been on the first examination. Towards the end of December last year it appears the child got whooping-cough. Dr. Fletcher does not appear to have seen the child until towards the end of that, but in January there was an occasional whoop. Towards the end of January it was found that the left lung was impaired above, dull at the base, especially behind, somewhat bronchial breathing at the base behind, feeble at the front, and there was a certain amount of scanty irregular râles, on the right side also there were some loose râles. At this time the child had a good deal of cough. After February there was less cough, and it was found that there was no decided râle; the breathing and the percussion were pretty much as noted in January. From that date until shortly before admission of the child to this hospital the patient seems to have gone on fairly well, but seems to have had little recurrent attacks of troublesome cough. About three weeks before admission it was believed there was some disturbance, as there was evidence of pleurisy, friction at the left base, and the temperature rose to 103°. The child was not admitted until this friction had passed off, but the general catarrhal condition remained. After being admitted here the child improved very decidedly. Its aspect was not really bad at first, but after admission it improved. The slight respiratory distress it had felt ceased, the frequency of respiration diminished, the temperature was not more than slightly above the normal, the position of the heart remained the same, and a few days after admission it was found that there was an improved note at the left apex, although it was more or less dull as we passed down to the base and posteriorly. There was very much less râle than when the child first came in, and the sounds of breathing generally were improved. There was faint breathing, slightly bronchial in character. On the right side also the movements were fair, the breathing sounds were fair, and there was great diminution in the moist sounds, indeed there were scarcely any at all. Ten days later, that is to say, yesterday, the child still continued to improve, and there was only an occasional spasmodic cough; the appearance of the chest was more normal, and one could not detect any very great difference in the movements of the two sides during breathing, and evidently the movement was better on the left side than it had been. The breathing was, however, nowhere natural on that side, but everywhere a little bronchial and catchy. There is an absence of any decided additional sounds. On the right side one may say the breathing is almost natural, at any rate you would not detect anything wrong.

Before I proceed to discuss that case I will describe another, which is at present under my care at the Chest Hospital. A child eight years old was admitted this month with a very troublesome cough, and a history of some small amount of blood-spitting for four days. You see that is an older child than the last. It was said to have had cough, and never to have been quite free from cough since the age of four months. It was said to have had five or six troublesome attacks of hæmoptysis in the last four or five years, the last of which happened one and a half years ago. The child had had measles and chicken-pox at dates unknown. The cough trouble could not be dated back to any particular trouble. There was a tubercular history on the father's side, both father

Digitized by Google

and grandfather having been tubercular; there was also a tubercular history on the mother's side; and there were said to be other children in the family who were not strong in the chest, but it is not definitely stated that any of those children were tubercular. This child on admission was somewhat delicate, fair of colour, weak looking, but moderately well nourished. There was a good deal of troublesome cough at first, but there was no spitting after the child was once admitted to the hospital, and there was certainly no bleeding. The temperature was 101° on the first day, respirations about 30, pulse 110, of fair volume; urine natural. With regard to physical signs, it was found that the resonance on the left side was deficient, the dulness being most marked at the apex, anterior and posterior, and the movements deficient. There were coarse moist râles and crepitations, rather creaky sounds, with irregular bronchial breathing at that side. On the right side there was fair movement, fair resonance, but the breathing sounds were harsh with rhonchal sounds and scattered râles, that is to say catarrhal sounds. The heart sounds were clear, the apex slightly displaced upwards and towards the left somewhat outside the nipple line. There was no night sweating. That is to say there were signs of general pulmonary catarrh, not symmetrical, with signs of contraction and deficient movement on one side, which may mean that there is commencing fibrosis, as in the other case.

After a few days in hospital this child's aspect improved considerably, and there was very much less cough. There never had been any sputum. The temperature had gradually subsided to normal, although there was a slight rise in the evening of about half a degree, but not more. In the lungs there was much less abnormal sound. There was improvement in the upper part; the apex which had been on the whole the most dull, was the more resonant now. There were some crepitant sounds to be heard. On the right side there was a little harsh breathing, and there were occasionally râles if the patient took a deep inspiration. Later on still, that is to say two or three days ago the general condition had still improved; there had been a gain in weight, very slight occasional cough, evening temperature still a little bit high, but almost normal. The left side shows an appearance somewhat of contraction and deficient movement; breathing everywhere on that side faint, irregular, slightly bronchial, but scarcely any of the added sounds. On the right side one might say the breathing and the movements were natural.

So you see the general condition in these two cases was somewhat similar, and the history is to a certain extent alike. Also the physical condition and the changes noted from time to time bear a resemblance. Cases of this kind are not infrequently to be met with. Usually the history of the starting point of the trouble is uncertain, and it is so in these cases, but there is a presumption that the trouble originates in measles or whooping-cough, or broncho-pneumonia, or possibly some pleurisy. It may be said that these children may suffer from occasional or even oft-repeated attacks of bronchitis or pulmonary catarrh; that in the intervals the condition is fairly good, and the nutrition, on the whole, well maintained, and that even during the attacks, although the child may be pulled down a great deal, it does not waste very decidedly, and often it does not lose appetite very much. Sometimes the temperature is scarcely raised at all, and frequently there is no sweating. Very often it would seem that the ordinary course is for the child to recover, and pass back to its normal condition, which might be said to be one of general good health.

On seeing a child of this kind for the first time it is perhaps natural to make the diagnosis of tubercle; although when you get more particulars of the history and you have an opportunity of further observing the case, you do not feel so much inclined to admit this view; still I do not think it will be found altogether easy to exclude it. In fact there are many cases in which you cannot be certain in saying that you can exclude it, although your opinion may be against it. I think, indeed, that many of these cases ultimately become affected with tubercle, and that when this occurs the children succumb pretty rapidly.

As against tubercle to begin with, there are the general points which I have mentioned, more especially the fairly good maintenance of the nutrition through the periods of febrile disturbance, the paroxysmal character of the cough, with occasional irregular expectoration, which in young children in these cases is certainly much commoner than in young children generally, the sputum being very frequently swallowed. It is only in the

Digitized by Google

cases where the sputum comes rather in volleys that you are likely to get expectoration, as you do sometimes.

Then another point is the absence of bacilli. As I have said, often you cannot get the sputum to examine. But in such cases as can be examined no bacilli, for the most part, have been found. There is a very rapid variation in the physical signs, variations which you may note while the patient is under observation, which are much more rapid than in ordinary cases of tubercular infiltration. These physical signs are collapse in varying degree, contraction, deficient movement, and auscultatory signs which may vary from nothing more than slight catarrhal sounds up to almost anything,—that is to say, the sounds which you get in varied conditions, such as pneumonic conditions, broncho-pneumonia, contraction, breaking down, irregularities of lung movement, cavities—almost every possible variety. A temporary partial, or possibly more or less complete unilateral collapse of lung in children seems to be common enough, and to be often completely recovered from without obvious traces being left behind. Sometimes, where you have every reason to believe that there has been a patch of pneumonia in a young child from febrile disturbance, and so on, you may get no physical signs which will justify you in going beyond the diagnosis of collapse over a portion of the lung, or almost the whole of it. That may last for a time and then pass away, and there will be complete recovery. You may get the same partial collapse apparently attributable to bronchial tube-blocking. Sometimes there is slight effusion, but an effusion which does not seem to be enough to cause the amount of collapse of lung which occurs. It is not a mere collapse due to pressure. Sometimes this collapse is so marked, and persists for such a time, that the lateral curvature which is the result has been mistaken for the result of spinal bone disease.

I cannot find any specimen in the museum which one can exactly associate with the kind of case which I have been describing to you, and I do not think it is surprising, because these children seem to live, or, at any rate, they do not die off rapidly. They seem to get recurrent attacks of acute trouble with cough, and they may be frequently in the hospital. I think it is always rather difficult to get post-mortems in such cases, as very likely they do not die in hospital, but usually get rapidly well, and when the fatal attack comes, perhaps the patient is not in hospital. One is rather driven to simply showing you other cases of permanent contractions following on chronic changes in the lungs and pleura. There are specimens on the table of that kind. Here is a specimen of a puckered and contracted lung, a localised pleuritic inflammation. There seems to have been no tubercular disease. Here is another case of chronic phthisis, with shrinking and fibrous change of the lung, a positive cavity, and also thickened bronchi, closely packed, and in some cases dilated. Here is another case of fibroid change with dilated bronchi, not supposed to be tubercular. Here is another of thickened pleura with dilated bronchi and surrounding fibrosis, but no evidence of tubercle. Here is another case of chronic empyema and contracted lung, with adhesions and contractions on that side. Here is a remarkable case showing contraction, the specimen consisting of the thoracic viscera from a case of chronic phthisis. The anterior surface shows the very great size of the right lung, which is distended. The shrunken left lung is covered with much thickened and roughened pleura. The heart, the base of which is considerably drawn to the left, is also altered in position on account of pericardial adhesions. You see the left lung is practically destroyed and shrunken, and undergoing fibrosis; the upper lobe is occupied by a large smooth cavity. The lower lobe also has a cavity. This is no doubt tubercular, but I show it to you because of the contraction and fibrosis of the left lung and the displacement of the heart.

So you see one has, in these specimens, cases of chronic phthisis with cavities and fibrosis, cases of chronic pleurisy with fibrosis extending into the lung, thickening and fibrosis round the bronchi, with, it may be, dilatations of the bronchi and the results of chronic pleurisy, where there has been also empyema. All these in adults and young people may lead to great contraction of one side. It is my belief that all the cases, such as I have described, follow on some pleural or lung lesion supervening upon broncho-pneumonia or pleurisy leading to fibrosis of the lung, irregular contraction in parts, and dilatation, sometimes great dilatation in other parts of the bronchi.

Digitized by Google

The cause of the rapid variation in the physical signs may be considered, because it is very remarkable how you may, within a period of two or three weeks, find a child with auscultatory signs which would make you think there are irregular cavities all over, and consolidations, and altogether no natural sound whatever, yet all that may clear up, and you may have, not proper breathing it is true, but not much abnormal sound. I suppose there is catarrh, and the irregular lung gets in part blocked. The variation of the balance of muco-pus formation leads to plugging of tubes instead of its easy regular unnoticed discharge, which may lead to collapse of the dilated tubes beyond, and the filling up of such dilated tubes sometimes with thick mucus; sometimes there is a rupture of such tubes, and an increase in the amount of air-containing space.

The special point which I would call your attention to about these cases is this, from the clinical point of view. Do not be too ready to make the diagnosis of phthisis, or to express the opinion that there will be a rapid termination in such cases. After you have had a little time for observation of the case, it is very likely that a good prognosis for at least the immediate future may be given. Of course, naturally every catarrhal attack is a danger, and such children are unlikely to make a good fight when they are attacked by anything acute. But it is remarkable how these patients do get over such attacks as I have described, and they may appear little worse after each one, and they often go on and live for some years. As far as one's experience goes, they either rapidly succumb to some acute attack, or else they rapidly get well. I think the cases for which one may give the most favourable prognosis are those where in the intervals the abnormal sounds quiet down to the least possible, that is to say, to poor breath-sounds, or only slightly bronchial breath-sounds, with slight irregularities or variations from point to point, but nowhere any decidedly unnatural sound, rather a lack of good sound than any great abnormality. The least favourable cases I regard as those where there remain doubtful cavity-sounds, irregular vocal resonance, or slightly amphoric breathing, or, of course, anything in the nature of gurgling sound. In such one is disposed to say the prognosis is less favourable, particularly if the recurrent attacks of expectoration are frequent, where the patient is suddenly troubled, and discharges a considerable quantity of sputum, which comes as it were in a volley, and especially if there be any fœtor. In one of these cases which I have mentioned there was a time when there was a sudden expectoration in large quantity, but I think it may be doubted whether in that case there was not at one time a definite pulmonary abscess, or possibly an empyema which suddenly opened. But the special point I would again mention is, do not be too ready to pronounce such a child tubercular. Such a pronouncement may affect you in private practice very unfavourably. Such a child may ultimately die of tubercular disease, but many such children live for a considerable time, notwithstanding that they are generally in an unstable condition.

The Effect of Alcohol on Uric Acid Excretion.—R. Roseman mentions various other investigations of this question, which have had rather inconclusive results. A research of his own was carried out on a healthy adult unaccustomed to the drug. The experiment extended over a period of thirty days, first ten days under normal conditions, then ten days, during which large amounts of water were drunk, with the idea of discounting any objection that a possible subsequent increased elimination of uric acid might be due to diuresis produced by the alcohol, and finally ten days, during which 75 c.c. of alcohol were taken daily in 1500 c.c. of water. This dose at first produced distinct symptoms of intoxication, but tolerance was soon established. The results showed that the average daily amount of uric acid excreted during the normal and water periods did not differ appreciably from that eliminated on the alcohol diet. In the course of another experiment, conducted on a fasting individual, there was observed a total absence of uric acid from the urine for a period of two hours, a hitherto unrecorded observation on human urine. This was perhaps, however, due only to idiosyncrasy, but a distinct increase in the amount excreted was not obtained in either set of observations, and it seems that alcohol does not very greatly affect the amount of uric acid disposed of. Although this conclusion is at variance with the prevalent belief that gouty subjects, on an alcoholic diet, suffer an increase in the amount of uric acid given off, the author believes the problem is too complicated to be solved by a single set of analyses, and that clinical experience is the more reliable guide.—*Medical Record*, August 31st.

Digitized by Google

MECHANICAL OR TRAUMATIC ARTHRITIS.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

GENTLEMEN,—I propose considering with you to-day the varying effects upon the anatomy and physiology of a joint which are produced by the transmission of an excessive or abnormal force through it.

I will deal in the first instance with the transmission *on a single occasion* of an amount of force through a joint in excess of what it can sustain without undergoing change, and I would exclude cases of damage to a movable articular fibro-cartilage, or of separation of a portion of articular cartilage as the result of direct impact.

I must again call your attention to the fact that *the mode of reaction of the joint to injury varies largely with the age and vitality of the individual.* In a young subject the articular cartilage may be very severely bruised, and a certain amount of destruction may ensue in consequence, but if the joint be kept at rest for a time sufficient to allow of its repair it will show no very obvious change after a few months have elapsed.

Still, even in the comparatively young adult, articular cartilage may not be repaired if it has been sufficiently damaged by the injury, or if after it has been severely bruised it has not been kept at rest. Under these circumstances the bone exposed by the removal of the cartilage may become sclerosed and eburnated, and changes somewhat similar to those that take place after middle life may ensue. Occasionally, in the young subject, a peculiar overgrowth of cartilage and bone may arise in consequence of damage to a joint, so that the area of the articular surfaces is increased by the irregular deposit of bone and cartilage on its margins, while the range of movement is limited in a corresponding manner. This condition is fortunately uncommon.

We must, however, bear in mind that in young life the transmission through a joint of an excessive amount of force sufficient to bruise the articular cartilage, or the very vascular osseous tissue beneath it or in the growing line, provides a nidus of poor resisting power in these structures or in the inflamed synovial membrane, which is very likely to afford a foothold for the tubercle bacillus; and I take it that in joints some such local depreciation is almost always the necessary antecedent to their invasion by these organisms. On these grounds, any injury to a joint in young life should be met promptly by placing the part completely at rest, and if there is any tubercular history in the family, and especially if there is or has been any tubercular manifestation elsewhere in the body, germicides, as mercury, arsenic, and the iodides, should be administered freely for a time, till all suspicion of evidence of the effect of traumatism has completely disappeared. Even with medical men one has too often known the advice given, that the child should be kept on the move and the pain walked off or worn off by vigorous massage with disastrous results. After the epiphysial lines have become obliterated there is not always the same objection to exercise and massage, but even then it should be employed only when the surgeon has a full knowledge of the nature of the injury the part has sustained, and of the physique of the individual, and it must not be ordered indiscriminately, as is too often the case at the present time.

After a certain period of life, which varies within wide limits with the vigour and vitality of the individual, the joint behaves in quite a different manner as the result of injury.

Amongst people who lead an ordinary life this transition period varies usually between forty and forty-five or fifty, but amongst the labouring classes it antedates this age considerably in a degree proportional to the wear and tear to which the individual has been exposed.

In such an injury the bruised cartilage is not replaced, but is instead gradually removed, leaving an excavated surface of articular cartilage. If the injury is sufficiently severe, the whole thickness of the articular cartilage is absorbed at the point of impact and the layer of bone beneath it is exposed. The extent of the articular area through which the force was transmitted in the first instance may be small as compared to the total area of the joint surface, but the friction of the bruised area upon that which surrounds it on the opposing bone in certain movements of the part produces progressive spreading changes in both, so that in time the frictional destructive process extends to the

Digitized by Google

entire joint. Besides the sclerosis and eburnation of the articular surfaces there is often an excessive secretion from the synovial membrane, which becomes chronically inflamed, thickened, and bulky. Also, lips or edges of bone form on the margins of the articular surfaces, with the object of rendering the failing security of the joint a less marked feature.

Pieces of bone may be developed also in the synovial fringes and ligaments, and on the bottom of cavities as the olecranon and coronoid fossæ. These all tend to limit the range of movement of the articulation, though they increase its security within that range.

In the case of amphiarthrodial joints the removal of the bruised interarticular cartilage often results in the ankylosis of the bones to one another and in the obliteration of the joint. All these pathological changes, and indeed all those I am about to describe, have been regarded by pathologists as evidence of a disease called sometimes "osteo-arthritis" and sometimes "rheumatoid arthritis." The term rheumatoid arthritis is now usually confined to a specific disease whose clinical symptoms, though very variable in the several decades of life, are thoroughly well recognised. It has nothing in common with the conditions I am referring to here.

Fourteen years have elapsed since I communicated to the Pathological Society a paper entitled "The Causation and Pathology of the so-called Disease Rheumatoid or Osteo-Arthritis, and of Senile Changes."*

I pointed out that all the morbid conditions which were said to be manifestations of osteo-arthritis were produced mechanically and in obedience to simple general laws.

I could not find in our museums specimens of so-called rheumatoid arthritis which were not mechanical in their origin, or which bore any direct causal relationship to the disease rheumatoid arthritis as we know it clinically in the present day, other than such as are consequent on a limitation of movement, with a consequent specialisation of pressure.

I showed that precisely similar changes occur in the skeletons of domestic and other animals, and that they are brought about in the same manner.

Of the pathology of true rheumatoid arthritis we know little more than enough to say that it resembles in no manner the pathological conditions to which the same term is still almost universally applied.

The designation traumatic or mechanical arthritis, which I have adopted suggests the mode of causation of these changes, and, being fully descriptive, it appears to me to be both serviceable and scientific.

I will now proceed to illustrate, by clinical history and by pathological specimens, several typical examples of this variety of mechanical or traumatic arthritis, which is the term I employ to comprise all these results of injuries to joints.

1. An elderly woman trips over a piece of orange peel, and falls heavily upon the extended palm. The lower end of the radius resists the breaking strain which it sustains, and the whole of the force which is transmitted along the shaft of the radius

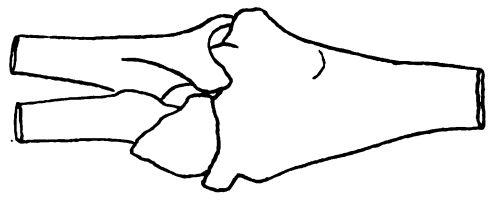


Fig. 1.

is impacted upon the radial head of the humerus, bruising the opposing surfaces of articular cartilage severely. After the accident the elbow-joint becomes painful, the joint remaining more or less completely fixed in the position in which it sustained the injury. After a time the tenderness in the part subsides, and the patient acquires some degree of movement, chiefly in the direction of rotation of the shaft of the radius, the range of flexion of the elbow-joint continuing very limited. On examining the joint the head of the radius is found to be much altered in shape, and the radial head of the humerus presents much bony deposit upon its margins. The styloid process of the radius occupies an abnormal relationship to that of the styloid process of the ulna, being on a higher level than on the opposite side. The pathological condition shows a destruction of the articular cartilage covering the opposing surfaces of the radius and humerus with frictional changes and sclerosis of the exposed bones. A quantity of new bone is deposited around the head of the radius, and about

* 'Transactions of the Pathological Society,' 1887.

Digitized by Google

the altered and limited facet on the humerus. A certain slight amount of change of a similar nature has taken place in the humero-ulnar and superior radio-ulnar joints, but this is due to extension from the radio-humeral segment in a simple and mechanical manner. Fig. 1 represents diagrammatically

FIG. 2 represents the anterior surface of the elbow-joint, which shows the several structural changes which result from the transmission of great force through it on a single occasion. (From *The Practitioner*, by permission.)

the changes which such a joint undergoes, while Figs. 2 and 3 are photographs of the same specimen.

This specimen illustrates very well the relative amounts of force transmitted to the humerus directly through the radius, and indirectly through the inter-osseous membrane and ulna.

2. A man, past middle age, falls heavily upon his side, striking the great trochanter of the femur with much force. In consequence of this injury he experiences great local pain in the hip-joint, aggravated by movement of it. If the legs are measured, they are found to be of equal length. The patient notices that after the injury his leg s not so reliable as it was before, that the movement of complete flexion of the hip-joint is accom-

FIG. 3.—Lateral view. (From *The Practitioner*, by permission.)

panied with discomfort at first, and later with pain, so much so that any movement of the joint is avoided as much as possible. He also suffers discomfort or pain in the joint in damp weather or during the cold east winds. I believe it is the similarity of this pain to that felt by rheumatic patients that has led clinical observers to include this traumatic condition among those classified under the old headings of "chronic rheumatic arthritis," "rheumatoid arthritis," etc.

Digitized by Google

I need hardly point out that after an injury to a bone, such as fracture, etc., the sufferer often complains of the same kind of pain under the same atmospheric conditions. After a time the patient notices that the damaged leg has become shorter than its fellow, and actual measurement shows it to be the case. This shortening increases during the lifetime of the patient, if he continues to throw his weight upon the articulation. The joint becomes progressively less secure, until at length, in many cases, the weight of the body cannot be supported by it at all. Soon after the receipt of the injury grating can be felt on flexion or extension, and this sensation becomes more marked as time goes on. If such a joint be examined after death, the head of the femur and the acetabulum are found to be completely altered in form and character. The opposing surfaces of the

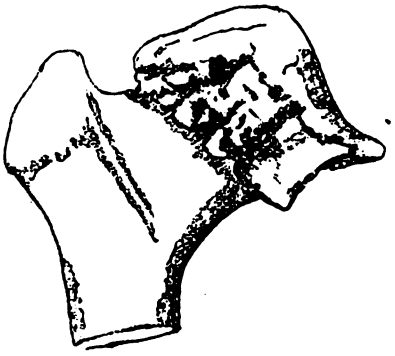


FIG. 4.

innominate bone and femur are rubbed down, sclerosed, and eburnated, while around these altered surfaces there is an abundant deposit of more or less dense bone, whose obvious function is to compensate for the alteration in the direction in which force is transmitted through the joint, and to render it as secure as possible under the circumstances. The capsule has become thin and lax, and has ceased to perform most of its normal functions. The synovial membrane in the joint is bulky, and contains an abnormal amount of synovia. Figs. 4 and 5 represent the condition and its mode of production from the normal state. The opposing surfaces of articular cartilage have been bruised sufficiently to interfere with their vitality, and there being no means of replacing the damaged tissue it is removed. The habitual transmission of force through the damaged joint results in the progressive destruction of the remainder of the articular cartilage, in the exposure, eburnation, and the gradual removal of the subjacent articular lamella of bone. These changes are followed by the others already described, the factors determining their evolution being solely mechanical. This all seems very simple and obvious to you. Yet at the time I wrote the paper already referred to in the 'Transactions' of the Pathological Society, this condition was, and, for all I know, may still be described in a well-known text book of surgery as "interstitial absorption of the neck of the femur." Such a pathology is founded on imagination and not on fact.

3. The following serves as another instance of chronic traumatic arthritis. A man falls heavily on the outer surface of his shoulder. He suffers much pain at the time, and he can neither bear

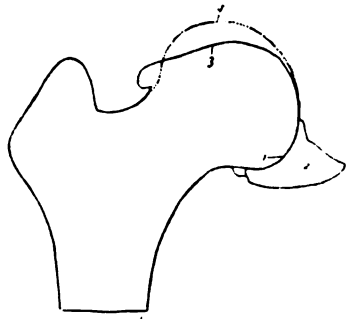


FIG. 5.

the shoulder-joint to be moved, nor can he perform any active movement himself, on account of the pain associated with it. If the shoulder be examined no fracture can be made out, and the outlines of the humerus and of the glenoid cavity are normal. The symptoms progress as in the case of the hip. The shoulder becoming flattened, the patient is unable to abduct the humerus, and the movements of the shoulder-joint become limited finally to a moderate amount of flexion and extension. On examining the joint after death, the opposing surfaces of the head of the humerus and of the glenoid cavity are found to be deprived of cartilage, the articular layer of bone is rubbed down and eburnated, and the surface of the humerus is rendered flat or but slightly convex, being much increased in breadth by the deposit of bone upon its margin. The articular surface of the scapula

Digitized by Google

has undergone similar changes. The synovial membrane is bulky, and the muscles of the shoulder, especially the abductor muscles, are much wasted. Here, again, the causation is quite simple. Force is applied to the outer surface of the humerus in such a direction that its articular surface is driven forcibly against that of the scapula, at right angles to the surface of the latter. Changes ensue in the manner already described in the hip.

Fig. 6 represents the shoulder-joint of a cabman who fell off his cab and pitched on the outer aspect of his shoulder. It exhibits the condition just described.

I would instance one more very common variety of traumatic arthritis in this joint. An elderly man receives a heavy blow, or pitches heavily on the point of his shoulder, breaking his acromion

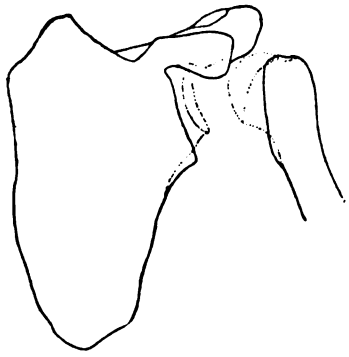


Fig. 6.

process, whose loose fragment is driven forcibly downwards upon the upper surface of the adducted humerus, bruising its articular surface severely, together with the intervening capsule and tendon of the supra-spinatus. Degenerative changes ensue in the contused portion of the articular cartilage, and gradually extend to more or less of the remainder of the cartilage of the head of the humerus and to that of the glenoid cavity. The upper portion of the capsule, with more or less of the tendon of the supra-spinatus, is gradually destroyed, and the humerus comes sooner or later into direct contact with the movable fragment of the acromion. The constant movement of these bones upon one another results in further changes in both, the acromial portion becoming excavated, eburnated, and lipped. The intra-capsular portion of the biceps tendon is destroyed, the long head acquiring an attachment to the altered great tuberosity. Other changes develop at the same time in the synovial membrane. I would call particular attention to this last injury, as it bears in an important manner on the elucidation of the causation and pathology of chronic traumatic arthritis. I showed that fracture of the acromion is of very common occurrence, and that the supposed rarity of the fracture depends upon a want of skill in recognising its existence. Also, non-union is due to the fact that unless the accident is diagnosed, which it very rarely is, or unless it be accompanied by some more severe injury, as fracture of the upper end of the humerus, the shoulder is not kept at rest for a sufficient time to allow of the acromial fragments uniting by bone, and, as a consequence, a false joint forms. When the blow which produced the fracture was sufficiently severe to contuse the articular cartilage of the head of the humerus and the intervening soft parts, the changes I have just described follow.

Anatomists are largely responsible for the non-recognition of these fractures, since they have chosen to regard the resulting ununited conditions as examples of non-union of an epiphysis, and, so far as I can see, upon no reliable evidence whatever.

In looking over some old letters the other day I came across one from the late Sir John Struthers bearing the date of November 30th, 1896, and referring to this subject. I make no apology for quoting a few lines from it, as his reputation as an anatomist was of the very highest order.

"I have to thank you for having led me to doubt the epiphysis theory for separate acromion process. It requires a considerable exercise of open mind to part with a long-held favourite theory, and I dare say that paper of mine at the beginning may show traces of the old impression, but the severe examination of all the evidence to which your destructive criticism led me sufficed to convert me without hesitation to the fracture explanation, notwithstanding the great family likeness of my specimens and the occasional occurrence of it in both shoulders."

Surgeons have readily accepted the explanation of the anatomists, apparently without investigating them. They have, however, observed the frequent association of this so-called ununited epiphysis with the presence of chronic rheumatoid changes in the shoulder-joint, and have explained the cause

Digitized by Google

of their association in various ways. One has gone so far as to assert that in cases of chronic rheumatoid arthritis of the shoulder-joint, a disunion of the acromial epiphysis from the rest of the bone takes place late in life. Such a statement hardly calls for criticism, and I only mention it to emphasise the frequency with which those changes which the pathologist regards as characteristic of the disease rheumatoid arthritis of the shoulder-joint are found associated with ununited fracture of the acromion. Can anything be more obvious and simple than the mode of development of an ununited fracture of this process of the bone, if its position and muscular attachments are considered, together with the difficulty of retaining the broken fragments at rest for any length of time, even if the fracture be recognised? The only two examples of bony union which I have observed in fracture of the acromion occurred in cases in which there existed in addition a severe comminuted fracture of the upper end of the humerus. Owing to the pain resulting from the presence of the latter fracture, the shoulder was kept at rest sufficiently to allow of bony union of the acromial fragment. Though I might multiply such instances of traumatic arthritis almost indefinitely, I think I have said enough to show that a severe injury to a joint is liable to be followed by the development of such pathological conditions as surgeons are still accustomed to regard as evidence of the presence of rheumatoid arthritis.

(*To be concluded.*)

GROSSE reports the case of a girl of five, whose right leg, especially below the knee, was markedly smaller than the left, even at birth. Nor did the right leg grow below the knee. Various different supports had been used without effect. Röntgen photographs showed the absence of the tibia, the fibula being alone. Prof. von Bramann operated, bringing the fibula into the knee-joint, taking great care that the epiphyseal cartilage was not injured. Foot and leg were then put in plaster. The former shortening of $5\frac{3}{4}$ cm. has been decreased 2 cm. She can now support herself without any splint, walking with a slight limp.—*Philadelphia Med. Journ.*, August 24th, 1901.

TWO CLINICAL LECTURES

ON THE

CONTROL OF ARTERIAL HÆMORRHAGE AND THE TREATMENT OF ANEURYSM.

Delivered at the Post-Graduate College,
West London Hospital,

By W. McADAM ECCLES, M.S., F.R.C.S.,

Senior Assistant Surgeon to the Hospital; Assistant Surgeon, City of London Truss Society, etc.

LECTURE II.

GENTLEMEN,—You will remember that in the preceding lecture we were discussing the question of the control of arterial hæmorrhage from a particular artery, and to-day I propose to deal with some other arteries before I say a few words about the subject of the treatment of aneurysm. With regard to arterial hæmorrhage from a punctured wound of the middle of the thigh, it is interesting to note that males are the chief sufferers, probably because when a sharp-pointed instrument happens to fall, a male instinctively tends to adduct the thighs and in this way a punctured wound is produced at the spot where the instrument may happen to impinge upon the thigh. On the other hand it is not a very frequent accident to occur in females, because they have a tendency "to make a lap" in order to catch any article that may happen to fall rather than adduct the thigh. I have never seen a punctured wound of the thigh occurring in a female from this particular form of accident. The first point in the arrest of bleeding from a punctured wound in the middle of the thigh is to control the hæmorrhage by pressure on the actual bleeding spot. Then apply a tourniquet, of which I show you several varieties here on the table. Petit's tourniquet is not much used in the present day for controlling arterial hæmorrhage, although it was once a favourite. If one has it near by, probably the most simple instrument to use is a rubber cord. A band of rubber is passed round the thigh and then, while on the stretch, it is brought into a narrow slit in a piece of boxwood, and so held in place. If there is no actual hæmorrhage at the time when the patient is seen, the difficulty then is to know what should be done whether the wound should be explored, or whether

Digitized by Google

temporary measures should be adopted in the hope that no very large vessel has been injured. It is the usual rule that if no hæmorrhage is occurring at the time, the parts should be thoroughly cleansed, dressed antiseptically, and a tourniquet placed in position, but not pressed, upon the main artery; some one in attendance should be instructed how to immediately apply the tourniquet if hæmorrhage should recur. On the other hand, if hæmorrhage is actually in progress at the time of the visit to the patient, there can be no doubt that the proper treatment is to cut down upon the vessel and tie the wounded artery on both sides of the aperture, pressure meanwhile being maintained upon the main vessel on the proximal side.

Sometimes the intercostal arteries, either from stabs in the thorax or from operations, give rise to very severe hæmorrhage; occasionally the same occurs after fracture of the ribs. The reason that the hæmorrhage is usually large is because the vessel cannot contract and retract properly, seeing that it is lying in somewhat dense tissues in the subcostal groove. A temporary means of arrest in such cases where there is a wound is obtained by passing in between the two ribs a finger-like pouch of antiseptic gauze and introducing into the interior of this pouch some strips of gauze. Then by drawing on the projecting portion of the pouch the artery can be compressed against the rib. This plan has been very satisfactory in a number of cases in which it has been employed. The deep epigastric artery is another artery which sometimes gives rise to troublesome hæmorrhage. The proper treatment is undoubtedly to cut down upon the vessel and tie it. The arteries of the spermatic cord are also likely to cause severe hæmorrhage, especially after castration. They may sometimes slip from the ligature, if this has been imperfectly applied, and give rise to very great extra-peritoneal hæmorrhage; here, again, the only thing to do is to follow up the vessels and re-secure them.

Hæmorrhage from the middle meningeal artery also deserves notice. A fracture of the skull about the area which is known as the pterion is liable to cause a laceration of the anterior branch of this vessel, which is derived from the internal maxillary, one of the terminal branches of the external carotid. Hæmorrhage from this vessel causes pressure on the brain by a collection of blood-clot between the dura mater and the bone. The treatment is to trephine. Turn down a semicircular flap which carries its own blood-vessels with it, and then trephine over the pterion—which is practically, in the adult, one and a half inches directly behind the external angular process of the frontal bone. On removing the crown of bone blood-clot is seen, the dura mater having been pressed and pushed inwards, since the blood is between the bone and the dura mater. When the clot has been removed by the handle of the scalpel, or washed out, the anterior branch of the middle meningeal will be seen lying on, and partly in, the dura mater, but not within it; so that to pick up the vessel with a pair of pressure forceps is not generally possible, and therefore it is necessary to take a needle threaded with silk and pass it underneath the vessel and ligature it in that way. If this anterior branch of the middle meningeal happens to lie in a complete canal in the anterior inferior angle of the parietal bone, then the canal requires to be plugged either with some aseptic wax or a strand of catgut. Supposing that this fails to arrest the bleeding, it is imperative to tie the external carotid artery (in preference to tying the common carotid), and the proper site to apply the ligature on the external carotid artery is between the superior thyroid and the lingual branches, because if the ligature were placed below, that is to say on the proximal side of the superior thyroid branch, then blood could be brought by the anastomosis between the inferior thyroid and the superior thyroid, and thence to the wounded meningeal vessel. This is a fact which it is important to recognise, otherwise the ligature will be no good. The same point may be mentioned in connection with hæmorrhage from the tonsillar and from the lingual vessels. Time will not permit of any discussion of the question of secondary and reactionary hæmorrhage; I will consider at once that of the treatment of aneurysm.

Treatment of Aneurysm.

Aneurysms are generally divided into two classes—external and internal—the latter those occurring in the cavities of the trunk, and the former those occurring on the limbs or head or the neck. Internal aneurysms are still to a very great extent within the domain of the physician, and medical treatment is generally carried out under two chief headings, rest and the employment of certain drugs. More recently a trial has been given to the intro-

Digitized by Google

duction into the tissues of gelatine, and there are some cases where this seems to have caused an increase of the coagulability of the blood and so the deposit of laminated clot in the sac of the aneurysm.

Surgical treatment, on the other hand, employs very definite means, and in a large number of cases these lead to a complete cure. Surgical procedures may for our purposes be divided into three great classes. First of all pressure, second ligature, and thirdly the introduction of foreign substances into the sac. I shall take as an example the commonest form of an external aneurysmal sac, that of a sacculated aneurysm upon the popliteal artery.

Pressure in the Treatment of Aneurysm.

Pressure may be applied in one of four ways. First on the main artery on the proximal side of the sac, secondly on the main artery or its branches on the distal side of the sac, thirdly on the main artery on the proximal side together with the main artery or its branches on the distal side without pressure on the sac itself, and lastly pressure may be applied directly on the sac.

(1) *Pressure on the main artery on the proximal side.*—This is the most usual way in which pressure is applied for the cure of aneurysm. This pressure may be of two kinds, mechanical and digital. Mechanical pressure is applied by means of a mechanical apparatus known as a tourniquet, and there are various forms of tourniquets in use for this purpose; but experience has shown that the inanimate insensitive tourniquet is by no means so effective an instrument as the sensitive living fingers, and that the tourniquet may do a considerable amount of local harm which the fingers are very much less likely to cause.

First with regard to the special apparatus required for digital compression, consisting of the living fingers to start with. It has been suggested that three pieces of rubber tubing linked together might be used as a substitute for the fingers, and in one case at least, to my personal knowledge, this apparatus was efficiently employed. All the same the fingers of the living person are better than even these soft rubber tubes. There is usually required, in order to get an easy method of pressure, a weight in the form of a cylindrical bag containing shot. Next there is needed an apparatus for allowing this weight to hang from a pulley so that it may come down to press upon the fingers. Then as to the number of assistants required. Two persons at least are necessary, or the principal and one assistant, one for pressing on the vessel and the other for determining the effect of that pressure on the aneurysmal sac, and then after the principal has performed his period of pressure, the assistant will take his place and press upon the vessel, while the principal will now determine the effect upon the sac. If, however, it is possible to obtain more assistants than one, it certainly makes the work lighter and easier. The patient should be prepared for this digital compression. Supposing he has an aneurysm behind the right knee, the pressure will have to be applied on the femoral artery just as it is passing in front of the horizontal ramus of the os pubis, that is the place where the main vessel lies closest to the skin and is most conveniently pressed against the bone. The parts should be shaved, thoroughly cleansed, and treated with methylated spirit and fine starch and zinc oxide powder, for at least, I think, two days before the pressure is commenced, so as to render the skin less sensitive than it would be otherwise. The patient should also be ordered to bed for these two days, so that he may become accustomed to the restraint of lying in bed, and also that his circulation may become adapted to the horizontal position. When pressure is to be commenced the limb should be placed in the position of abduction, flexion and external rotation, with the knee slightly supported upon a pillow. The surgeon who is to press the vessel takes up his position on the same side as the aneurysm, applies three fingers over the artery, which will be found half way between the anterior superior spine of the ilium and the symphysis pubis. The object of using three fingers is that the pressure may be somewhat distributed over the vessel, in order that when two fingers are removed during the exchange of position between the principal and his assistant, the third finger may keep up the control upon the vessel while the assistant is placing his fingers in position. The assistant takes up his position and places one hand, usually the left, beneath the bend of the knee and feels the aneurysmal sac, and gauges the amount of pulsation in it. If the surgeon had to maintain pressure upon the artery by his own unaided muscular effort, his fingers would very soon become tired out, and he would not probably be able to

Digitized by Google

continue the pressure for longer than possibly half an hour. If, on the other hand, he is merely to keep his fingers in position, and the weight in the form of the "sausage" is brought down so as to just rest comfortably over his fingers, then the amount of force which he has to put forward is infinitesimal. The weight does the work through the operator's living fingers. The amount of weight that is necessary for occluding the vessel varies considerably, dependent to a very great extent upon the thickness of the subcutaneous tissues and the rigidity of the vessel; but, as a rule, it is very much less than would ordinarily be thought to be necessary. This is the method for applying digital pressure as is ordinarily carried out. At the first day's trial, probably the patient will not be able to stand above two hours' digital compression. On the second day he may be able to stand three times as much, and on the third day a very considerable amount more. However, if pressure on the main vessel for a period, in all, of seventy-two hours has produced little or no change upon the aneurysmal sac in the way of diminishing the pulsation, it is probable that digital compression is not going to be efficacious. Then arises the question, will the digital pressure have interfered with the probability of a good result from subsequent ligature, because, if so, it is not advisable in most cases to attempt digital pressure before ligature. The answer to this question is that digital pressure will not have produced harm in point of view of a subsequent ligature. The pressure is not applied upon the vessel at the site where subsequently the ligature will be placed. For instance, if the femoral artery has been compressed near the end of the external iliac, the ligature would be applied either in Scarpa's triangle or Hunter's canal, well away from the site where the pressure has been applied. On the other hand, the pressure will have established, and to a very considerable extent, the collateral circulation, and so will diminish in all probability the liability to gangrene after ligature of the main vessel. It is always easy to diminish the amount of blood going to a limb, but it is practically impossible to increase it.

(2) *Pressure on the main artery or its chie branches on the distal side of the sac.*—It is not advisable to apply distal pressure unless there is no vessel of any size coming off between the sac and the site where the pressure is applied. For instance, if pressure is applied on the right common carotid, that is to say, on the distal side of an aneurysmal sac in the innominate artery the blood will have to come through the sac in order to reach the right subclavian vessel, and therefore no good whatever will accrue as the result of applying such distal pressure. If, on the other hand, the aneurysm is situated at the lower end of the common carotid, and not upon the innominate vessel, then distal pressure on the distal part of the common carotid, that is to say close above the sac, will be a possible means of cure, because the blood has no longer to come through the sac to go to a vessel, the common carotid artery having no branches. Of course one method of curing an innominate aneurysm might be to apply pressure both on the common carotid and on the axillary artery at one and the same time, but obviously this would be very difficult if not impossible to keep up.

(3) *Pressure upon the main artery on the proximal side of the sac, together with pressure on the main vessel on the distal side.*—This is carried out by means of what is known as Esmarch's method. Supposing there is a politeal aneurysm, the patient would have to be put under an anæsthetic and the limb allowed to hang over the side of the couch. It is sometimes said that the limb should be elevated. This is an error. Blood is needed in the limb, and it should not be exsanguinated. Then take a rubber bandage, and with the limb still dependent, apply it with a considerable amount of pressure from the foot to just below the knee, so as to press all the blood that is in the lower part of the limb up towards the sac. The bandage is then carried, without exercising any pressure, over the popliteal space, that is to say over the site of the sac, and then with pressure again is brought up the thigh to the upper part, so that there is a bandage along the whole length of the limb with pressure below the knee, no pressure at the knee, but pressure again above the knee. In this way it is obvious that the circulation is stopped and the sac of the aneurysm is left filled with blood, with the result that there will be in a certain number of cases at least coagulation of the stationary blood in the sac, giving rise to what is sometimes called a passive clot, and this may cure the aneurysm. Pressure has to be kept up for at least three quarters of an hour, with the patient under an

Digitized by Google

anæsthetic. Unfortunately this method is by no means a certain one, either with regard to formation of clot, or even if the clot forms with regard to permanent cure of the aneurysm.

(4) *Pressure directly upon the sac itself.*—This is perhaps most commonly carried out in connection with subclavian aneurysms by means of the application of some leather splint, possibly with a spring connected with it; pressure is thus brought to bear directly on the sac. Another way of effecting this pressure in the popliteal space is to flex the leg upon the thigh. Unfortunately this method is seldom followed with success.

Ligature in the Treatment of Aneurysm.

Ligature is undoubtedly in the present days of aseptic surgery the most safe and probably the most effective method of treatment of an aneurysm. A ligature again may be applied in several positions; first of all on the proximal side of the sac on the main artery; secondly on the main artery on the distal side of the sac; thirdly on the chief branches on the distal side of the sac, and lastly both on the proximal and on the distal side of the sac. Although I have put this double ligature last, it is by no means least in importance. Ligature both on the proximal and distal side of the sac is becoming more and more the one which is carried out by surgeons. It is certainly the ideal method of treatment of an aneurysm, provided it can be accomplished without too much danger. The vessel with its sac (say the popliteal artery) is dissected down upon; the artery above the sac is first of all separated from the tissues and secured by an aseptic silk ligature. Then the vessel below, that is to say on the distal side, is also defined and ligated in the same way, and then the intervening portion of artery, together with the sac, is dissected out and removed entire. In this way the patient is rid completely of his aneurysmal sac.

(1) *Ligature on the proximal side alone.*—This may be applied either at some distance from the sac or close above the sac. The artery may be ligatured—in the case of a popliteal aneurysm—at the apex of Scarpa's triangle or in Hunter's canal, that is at some distance away from the sac, or the artery may be ligatured in the popliteal space close above the sac. The former operation, known as Hunter's operation, has been up till quite recent years considered to be the better of the two. The vessel is then ligatured where it is more likely to be healthy, where it is in its normal anatomical relations, where there is less likelihood of septic inflammation passing from the site of the ligature down to the sac, and lastly, where there will be a better collateral circulation, whereby a small stream of blood is brought into the sac, necessary for the formation of the clot. Hunter averred that the whole of the blood-stream was cut off from the sac. Fortunately this is not so. Otherwise no clot could be formed in the sac; at any rate, no active or laminated clot.

(2) *Ligature of the main vessel on the distal side of the sac.*—This is inexpedient except in certain parts of the body. Practically speaking the only place were distal ligature of the ordinary type is nowadays applied is upon the common carotid at the distal part for an aneurysmal sac, which is in the proximal portion of the same artery. On the other hand, in a certain number of cases a distal ligature is applied by an amputation of the limb; for instance, for a subclavian aneurysm the whole limb as high up as the shoulder-joint may be removed and the axillary artery tied.

Now with regard to the question of ligature of main branches. In the case of an innominate aneurysm it is of very little use, if any, to ligature the common carotid alone. The ligature must be applied both to the subclavian and to the carotid, but a ligature applied to both at one and the same time is a very serious matter, so that in the majority of cases this double ligature has been carried out at two operations.

Introduction of a Foreign Substance into the Sac in the Treatment of Aneurysm.

The first method to which I would like to draw your attention is one which is probably familiar to you all, that known as Macewen's. It is called needling. The method that Macewen suggested was the introduction of very long slender fine steel needles, and his method is of chief use in a sacculated aneurysm with a very small communication between the artery and the sac. The needle is introduced through aseptic skin, through the subcutaneous tissues, and through the wall of the aneurysm, and then made to impinge upon its opposite wall, the patient being under an anæsthetic. Then by reason of the pulsation of

Digitized by Google

the sac the end of the needle is rubbed against the inner surface of the sac wall so as to render that surface somewat roughened, and therefore to excite the deposit of clot upon that particular spot. Then withdrawing the needle a short way its point is made to impinge on another area of the interior of the sac, and thus by repeatedly allowing the needle to rest for a few minutes on many spots a very considerable portion of the interior of the sac wall can be acted upon by its point. The results of this method, particularly for certain internal aneurysms, as, for instance, the intra-abdominal, have been distinctly satisfactory at first but, unfortunately, relapses have occurred and the case has progressed to a fatal end, though life has been prolonged. There is, however, one danger in this method, and that is the question of hæmorrhage from the puncture. But if a very fine needle is used and only one puncture is made, and care is taken not to enlarge that puncture after the needle has been introduced, the loss of blood is very slight, if any. Of course the whole procedure must be undertaken with the strictest aseptic precautions. The second method is the introduction of wire—usually a large quantity of fine silver wire. Here, again, there have been some very satisfactory results in internal aneurysms. A very small trocar and cannula is introduced obliquely into the sac, so as to make a more or less valve-like puncture. The trocar is withdrawn and wire is passed through the cannula and slowly and gradually introduced into the sac. The amount of wire that is passed depends entirely, of course, on the size of the sac and the facility with which the wire can be introduced. Another method is the introduction of needles attached to the poles of a battery, and through the needles a current is passed so as to produce a certain amount of electrolysis, and thus clotting of blood takes place. This method has not given such satisfactory results as the two former. The last method is the introduction of styptics. Perchloride of iron, carbolic acid, and more recently liquefied gelatine have all been used. Styptics are, however, highly dangerous. There is much likelihood of their causing clots which are carried in the form of emboli into other vessels and give rise to serious mischief. There are cases on record where this method has been tried in the case of subclavian aneurysms, and clots have been carried to the brain with fatal results.

The treatment of aneurysm is certainly becoming more hopeful, partly on account of the fact that it is undertaken very much earlier in the disease, and partly because patients more readily undergo the methods which are employed by the surgeon than those employed by the physician, which are apparently often considered by patients as a species of cruelty.

TREATMENT OF CARBUNCLE.

General tonics, like quinine and iron, with large amounts of nourishing food are indicated. Opium or other anodynes may be required to relieve pain and procure rest. Stimulants should be given only when required.

In the early stage ten to twenty minims of a 5 or 10 per cent. solution of carbolic acid in glycerine may be injected into the central portion of the mass with the view of aborting the mischief. If seen later, firm compression by straps of adhesive plaster applied concentrically may be made, leaving the central orifice free for the discharge of sloughs and applying an antiseptic dressing over the straps.

Another plan, applicable in the early or late stage as well:—Place patient under an anæsthetic; freeze the parts to make them friable; make one long incision or several crucial incisions through the mass; remove all sloughs and decaying tissue with a sharp curette; disinfect, drain, and suture, as in an incised wound.

Another method of treatment is the application of warm, moist, antiseptic dressings, covered with thin rubber cloth or oiled silk, removing sloughs as soon as loosened, and using iodoform, aristol, europhen, or similar antiseptic powder freely. The use of poultices is harmful and should be avoided.—*Sajous' Annual and Analytical Cyclopædia of Practical Medicine.*

Digitized by Google

THE CLINICAL JOURNAL.

EDITED BY L. ELIOT CREASY.

No. 466. WEDNESDAY, OCTOBER 2, 1901. Vol. XVIII. No. 24.

CONTENTS.

PAGE

*A Clinical Lecture on the Incidence, Nature, and Treatment of Chorea. By Sir DYCE DUCKWORTH, M.D., LL.D., F.R.C.P. 369

*A Clinical Lecture on a Case of Recovery from Fracture-dislocation in the Upper Dorsal Region. By CHARLES A. MORTON, F.R.C.S.... ... 372

*Mechanical or Traumatic Arthritis. By W. ARBUTHNOT LANE, M.S. (*concluded*) 377

* *Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers,* 22½, *Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d*; *and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

CLINICAL LECTURE

ON

THE INCIDENCE, NATURE, AND TREATMENT OF CHOREA.

By Sir DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Physician to St. Bartholomew's Hospital, and Lecturer on Clinical Medicine.

GENTLEMEN,—I proceed to-day to discuss with you the various conditions which appear to determine the onset of chorea. I have already told you that the disorder alights upon the female sex in far larger proportion than upon males, and that most cases occur between the ages of five and fifteen.*

All modern writers allude to the frequency of rheumatism as an antecedent or concomitant of chorea. In this country, and in France, this connection is regarded as of greater significance than it is in Germany, but even now there is hardly any English writer on medicine who ventures to hold the extreme view that I maintain, viz. that chorea is a rheumatic disorder of the brain. The frequent association of endocarditis is well recognised, and no one is able to point out any specific differences between what has been termed choreic endocarditis and rheumatic endocarditis. I hold that it is impossible to do so. The idea that the disorder arises from cerebral embolism resulting from endocarditis is now no longer held. No other antecedent or concomitant disease figures so largely as rheumatism in cases of chorea, but other maladies have been recognised as spreading it, as scarlet fever, puerperal fever, gonorrhœa, and pyæmia, all, be it noted, infecting maladies. But in cases of chorea which have been associated with, or preceded by, such maladies, I think it far from improbable that there is a predisposition to rheumatism in the patient, and that, as happens certainly in the case of scarlet fever, the onset of that disease distinctly lays the patient open to rheumatic infection. We distinguish, of course, from this cases of scarlatinal arthritis, which are not rheu-

* 'CLINICAL JOURNAL,' July 31st, 1901.

Digitized by Google

matic, and are not seldom the result of mixed and pyæmic infection. Other conditions have also been noted as antecedents of chorea, as surgical operations, the presence of intestinal worms, adenoid growths in the pharynx, also defects of ocular accommodation. With respect to all of these, I would venture to suggest that the subject of chorea will be found to be of the rheumatic habit, and that all these alleged provocations are not the specific determinants of the disorder. Indeed, I have grave doubts about all of these as excitants of chorea, save the possibility of shock and emotion as connected with surgical operations.

We note that children are specially liable to chorea, and we know that in early life the nervous system generally is more susceptible and unstable than at a later period. Youth, then, *per se*, is a factor in this matter. Chorea is met with in certain families, and heredity counts for something in the case. It is well ascertained that a tendency to become the subject of rheumatism runs in certain families, and this is as marked as is the proclivity to tuberculosis in others, the tissues or soil being specially apt to react badly against invasion of rheumatic infection. Mental worry, excitement, educational pressure, and fright are all recognised as determinants of attacks of chorea. It has always been difficult to trace a connection between fright and the onset of chorea. I have never regarded this as difficult of explanation. The effects of fright in certain people, especially in children, are profound and very grave. I have known the most intense anæmia induced by fright in a very short time, and the condition here can only be a rapid spanæmia, or a depression of hæmoparesis, as a result of intense nervous influence. If any of you have ever undergone the agony of a severe fright, as I have, you would know how profound is the influence on the nervous system. The ordinary subjects of chorea are persons of a very mobile and unstable nervous system. The effect of fright is probably to lower the trophic rhythm of this system, and to lay it open to immediate invasion by any malign or infective influence that may be at hand, as a *locus minoris resistentiæ*. I would say, then, that in cases of chorea the brain is rendered by fright, or strain, or emotion, specially vulnerable and sensitive to the effects of rheumatic infection which alights on the brain-cells and neurons, instead of on the throat, the joints, or the different cardiac textures. Chorea, then, is for me rheumatism of the brain. I feel as certain of this as of anything I know in all medicine, and I have been convinced of this for many years. The recent researches of Drs. Poynton and Paine have now rendered this conception as nearly positive by way of demonstration as anything can be. These observers have induced chorea in the rabbit by the inoculation of rheumatic diplococci, and have detected the latter in the pia mater and endothelial cells of the capillaries, and dipping into the motor cortex of the brain.* In a case of fatal chorea they discovered the organisms in the mitral valve and the motor cortex. They conclude that chorea is induced either by the presence of these diplococci in the pia mater and the brain, or by the toxins generated by them. The morbid anatomy of chorea relates to hyperæmia, small hæmorrhages, and foci of necrosis in the brain, and to small emboli or thromboses in the terminal capillaries. These changes have been detected in past years by Dickinson, Charlewood Turner and others. Some of these have been aptly compared by Hughlings Jackson to those found in the cardiac valves and subcutaneous nodules as the result of rheumatic infection. The morbid anatomy of chorea, then, so far as it has been elucidated, shows nothing incompatible with the idea of an infective inflammatory process dependent on microbic invasion, or its toxic effects, and the symptoms of the disease appear to indicate that these changes are due to multiple local lesions, to deposition of particulate doses, rather than to any generally diffused toxæmia.

We are to expect, if all this be true, that the determination of the morbid process to the brain will only occur in persons specially predisposed by brain-weakness or textural instability, and the disorder may thus be mainly manifested as a rheumatism of the brain. We see often enough that other textures may be involved in the joints, skin, throat, or heart, in the course of any given case. Arthritis or carditis may precede, or accompany, or follow on the brain manifestations, thus furnishing the keynote of the case, if such be required. Our narrow ideas in the past have alone prevented our recognition of the full significance of these clinical facts. The vulgar idea that rheumatism is essentially

* 'Lancet,' May 4th, 1901.

Digitized by Google

and exclusively a disease of joints and of the heart has hitherto limited our field of vision, and our clinical conceptions of the disorder.

One argument has been strongly pressed against this view, viz. the fact that treatment by salicylates proves commonly ineffectual in subduing chorea. I am not so sure that this is really a fact, but if it were true, it would be but a feeble argument in the case. We are not justified in recognising salicylates as specific anti-rheumatic agents. And this leads me to discuss with you the therapeutic measures that are advisable in chorea or rheumatism of the brain.

In the patient whose case forms the text of these lectures, we have had to deal with chorea in its gravest form, that variety known as *chorea insaniens*. This intense form may arise in the course of any case. This child appeared maniacal for many days. Her power of speech was severely involved, and it was interesting to note that her right side was the more gravely affected, affording evidence of damage in the Rolandic area on the left side of the brain. The recovery here has been most satisfactory. You see that the child is now quite quiet, and free from all fidgets, save in the fingers of the right hand. She speaks clearly and fluently. The average duration of all cases of chorea is found to be about ten and a half weeks. The disorder here has now lasted for ten weeks, and we may regard it as practically at an end.

The treatment of chorea by drugs can hardly be considered as satisfactory. I spoke in my last lecture of the distinct value of chloral-hydrate as a remedy for inducing quietude and sleep in these cases. Paraldehyde has also been of much service in this instance. Many drugs have been credited with virtues in curtailing the malady. Zinc salts were much in vogue at one time. Arsenic has long had the best reputation, however. The late Dr. Farre, of this hospital, was of opinion that Valengin's solution, the acid preparation of arsenic, was more useful in chorea than Fowler's alkaline solution. It was urged some years ago by Dr. Wm. Murray, of Newcastle-on-Tyne, that the most rapid cure of chorea was to be witnessed if large doses of the drug were employed. This he learned from an irregular practitioner whom he attended on his death-bed, and who had enjoyed a reputation for rapidly curing this disease. Ten or twenty minims, or more, of Fowler's solution were advised, repeated thrice a day, and these large doses were declared to be well-borne. Many of us have tried this plan, with care, so as not to induce toxic symptoms, but I am compelled to say that I am not satisfied with its merits, and there is always a risk of inducing peripheral neuritis, which is not lightly to be disregarded. I suppose no remedy is more resorted to than arsenic in these cases. It is indicated as a general nervine and nutrient tonic, and it may, and probably does, possess some antitoxic properties. We recognise its value in malarial poisoning. I am disposed to think that we may find in the early stages of the disorder that silicylate of sodium with bromide of potassium is a good combination; and in the later stages that arsenic may be of service, together with iron. Antipyrine is also of value in the earlier stages. Rest and quiet are essential. It is well to carpet a ward in which a case is treated, to screen off the bed, and subdue the light. The nourishment should be good, and given by mouth or nasal tube according to circumstances. During recovery a little wine is useful. The late Dr. Radcliffe, of the Westminster Hospital, treated chorea by large doses of wine and morphia, but I do not recommend this practice. If sleep can be secured, and a sufficient amount of food be taken, most cases do well.

The varying degrees of severity of the malady probably depend on the amount or intensity of the rheumatic infecting agent, and the psychical and local motional symptoms are best explained by the implication of more or less brain substance in varying localities. It has been suggested by Drs. Poynton and Paine that the brain is rendered specially susceptible and vulnerable by a latent rheumatic process in these cases; that rheumatism produces a cerebral state analogous to that produced by fright, and that when fright acts upon a rheumatic brain, it reacts to it just as the rheumatic part reacts to strain, and becomes the seat of carditis. I would rather regard the brain in these cases as unduly sensitive and vulnerable, unstable from hereditary weakness, and being overwrought by excitement, fright, or pressure of education, is the selected texture on which the rheumatic process alights. And at a varying interval after this determination we may expect the onset of choreic symptoms. These patients require extreme care in their nurture and up-bringing, and any undue severity or hardship on the part of parents and

Digitized by Google

teachers is especially to be avoided. The disease may recur if the child be submitted to the determining causes of the primary attack, and a recurrence of chorea is simply a return of the rheumatic process to the brain.

I think I told you that chorea was a more dangerous disorder when it occurred at the age of puberty. We may find the explanation of this fact when we bear in mind that at that time there is a general perturbation and excitement of the nervous system, more marked indeed in the female than in the male, and therefore we may believe the brain to be specially susceptible and likely to suffer from rheumatic infection. We might well wish to see in such cases a metastasis from the brain to the joints, but we hardly meet in rheumatism with the metastasis that may sometimes be witnessed in gout. Lastly, let me recommend you to read Professor Trousseau's lecture on chorea in his 'Clinique Médicale.' You will there find that many of the views I have expressed in these lectures have been anticipated by that great clinician, but they have never received the attention they deserve.

The Role of the Lymph-glands in the Infectious Diseases.—In a thorough review of this subject (in the 'Bulletin Médical,' 1901, No. 39) Dr. Curtois-Suffit states that the lymph-glands physically, chemically, and anatomically represent differentiated organs for the protection of the organism against microbes and their toxins. According to the malignancy of the invading infection, the microbe may disappear before reaching the nearest lymph-gland; it may reach the nearest gland only; it may reach a far-off gland; or it may quickly reach the blood *viâ* the thoracic duct. It is most common, however, for the invading micro-organism to remain in the nearest lymph-gland, which enlarges. Polynuclear leucocytes arrive by the blood channels, and the lymph cells become detached. Far-off glands enlarge when the toxin or the microbe reaches them. These processes are most frequent in childhood, and may persist. The reaction may be so marked as to cause a general disturbance, glandular fever, or bronchial adenopathy. In adults the lymph-glands become sclerosed, and with old age they atrophy. These glands are then much less able to protect the organism.—*Philadelphia Med. Journ.*, August 31st.

A CLINICAL LECTURE
ON
A CASE OF RECOVERY FROM FRACTURE-DISLOCATION IN THE UPPER DORSAL REGION.

Delivered at the Bristol General Hospital.
By CHARLES A. MORTON, F.R.C.S.,
Professor of Surgery in University College, Bristol; Surgeon to the Bristol General Hospital, and to the Hospital for Children and Women.

GENTLEMEN,—Recovery from fracture-dislocation in the upper dorsal region is so rare, that I think this case is of considerable interest.

A man, æt. 21, was admitted to this hospital at 8 a.m. on August 24th last, with the history that when in the stooping position a sack of flour fell from a considerable height on to his shoulders. He could not stand afterwards, and was brought at once to the hospital, where it was ascertained that he had complete paralysis of the lower limbs, and anæsthesia to the level of his umbilicus.

I saw him first at 2 p.m. There was a distinct depression in the spine at the level of the second or third dorsal vertebra, the sternum was broken across the manubrium, and the left clavicle fractured. There was then no anæsthesia, but complete paralysis of both lower limbs. The patella tendon reflex was present on the left side only. There were very painful jerking movements of the left lower limb. There seemed to be action of the intercostals in respiration. The catheter had to be used to relieve retention.

On the following day (August 25th) there was a return of power in the right lower limb, and the patella tendon reflex had returned on this side. The bladder had to be emptied by catheter. The painful spasms in his left lower limb had ceased. A day or two later there was great difficulty in getting his bowels to act. Croton oil failed, but very frequently repeated doses of a saline aperient succeeded. At this time there was very marked tympanites.

By August the 28th the power of micturition had not returned. I then left home for my summer holiday, and did not see him again until September 16th. Unfortunately there are no notes of his condition during this period. At the date of my return I found motion and ordinary sensa-

Digitized by Google

tion in the lower limbs perfect. He got up two months after the accident. At first he could only walk by holding on to the furniture, and there was some spastic rigidity in the lower limbs, and exaggerated knee-jerks and ankle-clonus. Three months after the accident he could dispense with support, and could get about almost quickly, but some stiffness in the limbs persisted. The ankle-clonus had gone, but the exaggerated knee-jerks remained. Electrical treatment was carried out during this time. The gap in his spinous processes became less marked. He left the hospital at the end of November.

When seen again, on January the 2nd, his gait was still stiff and awkward. The deep reflexes were still much exaggerated on both sides, and there was some clonus of the quadriceps extensor on passive movement of the knee. He felt pain in the injured part of his back when manipulating his collar stud behind. Since that date he has married and returned to his business, and he now (June, 1901) walks so well that it is impossible, by watching him, to detect he has had anything the matter with his lower limbs. There is no rigidity or clonus present, but the patella tendon reflex on both sides is exaggerated still. The irregularity in the spine is still marked.

There was very distinct displacement of the vertebræ in this case, and hence no doubt that a fracture-dislocation had occurred—a fracture-dislocation rather than a pure dislocation, because a pure dislocation in the dorsal region is almost unknown. That the force which caused the fracture-dislocation of the spine was very great is shown by the fact, that it not only fractured the spine, but also the sternum and the clavicle. Probably the spine gave way, and then the pressure on the shoulders continuing, the sternum and clavicle broke, so that the sternum was fractured by indirect violence. Fracture of the sternum secondary to fracture-dislocation of the spine has been recorded in other cases, but it is rare. Probably the displacement of the vertebræ was much greater at the moment when the accident happened than when the patient came under observation, for in these injuries there is a tendency for the displaced vertebræ to recoil, into, or towards, their normal position, and the cord is very severely crushed at the time of the accident, and possibly compressed by some remaining displacement. We have here a specimen from the museum of dislocation of the lower cervical spine which shows with how little persisting displacement the cord may be hopelessly crushed. We know that this serious crushing occurs at the time of the accident, because in cases in which the force has only separated the vertebræ and then allowed them to fall back into their normal position, so that no displacement exists, we may, nevertheless, find the cord destroyed by the crush it sustained at the moment when the spine bent, and the connection of the vertebræ gave way or fracture allowed separation.

In this case though there was persistent displacement, and the cord had probably been damaged at the moment when the accident occurred, yet the symptoms of lesion of the cord began quickly to pass off. It is an interesting question then: What was the exact nature of the damage to the cord? It must remain uncertain whether the displacement of the vertebræ caused any permanent compression of the cord. Displacement of the vertebræ may exist, and yet, if the displacement is not very great, the cord will very likely escape compression. Mr. Bowlby in twenty-one cases which he examined post mortem found no compression by displaced bone.* The rapid recovery in this case is altogether opposed to the view that compression of the cord was present.

In some of these cases of fracture-dislocation, hæmorrhage occurs into the substance of the cord at the seat of injury (hæmatomyelia). Two views have been held as to the cause of this hæmorrhage. One is that the hæmorrhage is due to severe shaking of the cord in the spinal canal, and the other that it is due to actual stretching of the cord. The latter view is the one generally accepted. The hæmorrhage is usually quite localised, and at the region of the cord where such stretching is most likely to occur—the lower cervical region. If the hæmorrhage were due to severe shaking of the cord, probably the surface—and a large area of the surface—of the cord would be affected, whereas what is found in these cases is a central hæmorrhage at one definite spot. In such cases there may be no permanent displacement of the vertebræ; the partially separated vertebræ may have fallen back into their normal position again, but the symptoms of a trans-

* 'Brit. Med. Journ.,' 1890, vol. i, p. 1132.

Digitized by Google

verse lesion of the cord following immediately on the injury are very marked. And in such cases their may be two sets of symptoms, those due to actual destruction of the cord—a central lesion, and those due to compression of the surrounding cord structure by the central hæmorrhage. Thus, for instance, if such a hæmorrhage occurred at the level of the lesion in this case, we might get persistent paralysis and anæsthesia in the region of one or two of the upper dorsal nerves, and temporary paralysis and anæsthesia of the parts below, and this might be followed by a condition of spastic rigidity in the previously paralysed limbs. It would be difficult to say if one or two of the upper dorsal nerves were permanently paralysed, certainly there was no persistent zone of anæsthesia. But cases of hæmatomyelia without permanent displacement of the vertebræ are more frequently recovered from than cases of fracture-dislocation with complete crushing of the cord, and hence, although some displacement persisted in this case, yet the rapid recovery of the patient would lead one to suppose that possibly the lesion may have been of the nature of a central hæmorrhage rather than of actual crushing.

But there is another form of hæmorrhage due to spinal injury, hæmorrhage into the spinal canal, but not into the cord (hæmatorachis). This is described as occurring in one of three positions: (1) Between the dura mater and the vertebræ, on the posterior aspect of the dura mater, where so many veins are found in the loose connective tissue; (2) Between the dura mater and the arachnoid; (3) Between the arachnoid and the pia mater of the cord. In the hæmorrhage between the dura mater and vertebræ we might expect the hæmorrhage to be localised, and, if of sufficient amount, to exert serious compression on some particular segment of the cord. Such compression, however, seems to be very rare. In the other two forms of intra-spinal hæmorrhage such localised compression is also described, but it is very difficult to understand how it can occur. The cord lies so loosely within it sheath of dura mater that to me it seems almost inconceivable that the blood should be so confined to one particular level of the cord as to compress it. We may reasonably ask, what is there to confine it to one particular level? I cannot believe that it clots so rapidly that it has no time for general diffusion in the sub-arachnoid or subdural spaces. Moreover we know that such generally diffused hæmorrhage does occur within the spinal canal, and is, indeed, sometimes only a part of intra-cranial hæmorrhage, and I have not come across the record of a case which seem to me to prove the existence of a sub-dural or sub-arachnoid hæmorrhage definitely localised to a particular level of the cord, and the cause of symptoms of compression of the cord at that level.

The violent jerking movements of the left limb on the day of the accident are not easily accounted for. There was no reason to think an irritative lesion of the nerve-roots, or of the cord at the level of the lumbar enlargement on the left side, was present, and it is difficult to see how a lesion of the cord in the upper dorsal region could produce such symptoms on the day of the accident. It is also worth noting that the patellar tendon reflex was lost in the limb which first recovered. This is just the opposite of what we might expect, as you will see when I come to speak of the prognosis of these lesions. Why the anæsthesia only reached as high as the umbilicus is not clear. It had disappeared when I first saw him, but I believe the observation to have been carefully made.

Perhaps some would be inclined to say that with the very rapid recovery of sensation, and more gradual return of power, the cord could only have been shaken, not crushed or compressed or torn up by hæmorrhage, and they would, notwithstanding the persistent displacement of the vertebræ, label the case concussion of the spinal cord associated with fracture-dislocation of the vertebræ. But the term concussion of the spinal cord is getting less and less used, even when intended to imply also a certain amount of hæmorrhage on the surface of the cord. How can the cord, suspended as it is in the spinal canal, be concussed? Although in this case the anæsthesia and paralysis were so quickly recovered from, the persistence of a certain amount of spastic rigidity in the lower limbs indicated a definite organic lesion. But whether this was the usual crush of the cord of only moderate severity, or a hæmorrhage into the cord, or both combined, it does not seem possible to decide. The persistent spastic rigidity with increased reflexes was probably due to some descending degeneration in the lateral columns.

Recovery from fracture dislocation of the spine

Digitized by Google

with injury to the cord in the upper dorsal region is rare, though several cases have been recorded at this level and even higher up in the cervical region. One of the writers of 'Erichsen's Surgery' states* that he has known at least four cases "in which recovery occurred after fracture of the lower cervical vertebræ with paralysis, more or less complete, from the neck downwards," and that in one of these the neck was broken in a fall in kangaroo hunting, and the patient after being paralysed for eleven months made a fair recovery. Mr. Thorburn, in his book on the 'Surgery of the Spinal Cord,' published in 1899, although he records several cases of fracture-dislocation in the cervical and upper dorsal region, under observation in the Manchester Infirmary, does not refer to any case of recovery. Mr. Battle† records a case of recovery from fracture-dislocation in the upper dorsal region. There was complete loss of sensation and motion and of deep reflexes in the lower limbs, and paralysis of the bladder. The displacement of the vertebræ was partially reduced, under chloroform five days after the accident, but sensation had largely returned before this. One year after the accident he walked several miles with a stick.

Gerster‡ records a case of fracture-dislocation of the fifth cervical vertebra in a boy. There was complete paralysis of arms and legs, and of bladder and rectum. Extension was used. The condition of sensation is not referred to. He recovered, except for partial paralysis of ulnar nerve and ataxic gait on the same side.

Dr. Porter, at a meeting of the Pathological Society on January 17th, 1899, showed a specimen of fracture-dislocation of the cervical spine which occurred twenty years before death, from bending of the neck forwards, caused by the fall of a wall on the man. The cervical vertebræ were crushed together, and the second, third, and fourth were at right angles to the lower three. The spinal cord had been but little damaged, and the symptoms after the accident were more cerebral than spinal, so that this was a case of fracture-dislocation without any distinct evidence of injury to the cord; but the deformity of the spine was so great it seemed unlikely that the cord escaped stretching at the time of the accident.

In Mr. Jacobson's article on Fracture of the Spine in the 'System of Surgery,' by Holmes and Hulke,* is recorded the case of a young man who lived fifteen months after a fracture-dislocation of the fifth cervical vertebra, with complete crushing of the cord at that level, and therefore persisting paralysis of arms and legs.

And Hilton, in 'Rest and Pain,'† records the case of a young man who lived fourteen years after the onset of complete paralysis of all his limbs from a fracture of the fifth, sixth, and seventh cervical vertebræ. He used to copy engravings with a brush held between his teeth.

Dr. R. Abbe‡ records a case of fracture of the fifth cervical vertebra, which was clearly shown by a skiagram, and in which complete paralysis and partial anæsthesia below the neck was present, but recovery took place in six months so far that only some wrist-drop and feebleness of grasp of the hand remained.

An interesting question for our consideration is: Would the patient have been benefitted by early operation? There are two conditions in which operation might do good in cases of injury to the spine from violent bending—localised hæmorrhage into the spinal canal, but not into the cord itself, which might be dealt with by opening the spinal canal and removing the clot, and persistent compression of the cord by the displaced vertebræ, which could be relieved in part by cutting away the spine and laminæ of the vertebra which is displaced forwards. I say, in part, because we might still have the cord compressed by the body of the vertebra below. In a dislocation or fracture-dislocation of the spine, the upper part of the spine is almost always carried forwards so that the cord is crushed between the laminæ of the vertebra above and the body of the vertebra below, and as a rule the cord is so severely crushed at the moment of the accident that it is hopelessly damaged. The vertebræ usually recoil to some extent into their usual position, and hence the persistent displacement does not indicate the degree of displacement which was actually present at the moment when the force acted on the spine, and hence the removal of the persistent displacement by operation usually fails to restore the

* Tenth Edition, vol. i, p. 808.
† 'Lancet,' 1892, vol. i, p. 1242.
‡ 'Annals of Surgery,' vol. xxi, p. 49.

* Third Ed., 1883, vol. i, p. 665.
† Third Ed., p. 116.
‡ 'Med. Rev.,' New York, 1890, vol. lvii, p. 352.

Digitized by Google

function of the damaged cord, for though the persistent compression may be relieved by cutting away the displaced laminæ, the cord has been too severely crushed at the moment of the accident to recover. It is an unfortunate fact that a badly crushed cord cannot recover. But even if the cord has escaped a hopeless crush at the moment of the accident, and the damage to the cord consists in persistent compression only, the removal of the laminæ (laminectomy) will only remove part of the compressing bone. The posterior and upper edge of the body of the vertebra below the displaced one may still press seriously against it. If we merely remove the laminæ above we may, however, greatly relieve the compression, but there is always the possibility that we may not. Some surgeons have advised the removal of the projecting ridge formed by the body below as soon as the laminæ have been removed; but others have pointed out that to make such an attempt would probably do much more damage to the cord than would be done by the persistent compression by the ridge, for it lies immediately beneath the cord, and the cord almost covers the bodies of the vertebræ. I do not believe that the cord could be turned first to one side and then to another in order to get at the ridge without dividing nerve roots; and even if these were not very important (as in the middle dorsal region) and might be divided, so that we might slightly raise one side of the cord, the use of a chisel and mallet in this position would be an exceedingly risky proceeding.

Although we must conclude that in the great majority of cases of fracture-dislocation of the spine the cord is hopelessly crushed at the moment of the accident, yet in a few cases this may not be so, and it is therefore most important to determine, if possible by clinical signs, if this hopless complete transverse crush has occurred. The fact that may aid us is, that in this condition of complete transverse crush the deep reflexes are lost. The condition of the superficial reflexes is not reliable as a guide. If, therefore, the deep reflexes are present we may conclude that there has not been a complete transverse crush.

The opinions of most surgeons at the present time seem to be that the chance of benefit from operation in fracture-dislocation of the spine produced in the usual way by indirect violence is so small that the operation is really hardly worth undertaking. No doubt the risk is not great, and the condition without operation is almost hopeless, yet the chance of doing good is so remote we can hardly advise it. I am not speaking of those very rare cases of direct injury in which a spine or laminæ may be driven in on the cord, and no displacement of the bodies exists; in such cases laminectomy is strongly indicated. The case that I have taken as the subject of this lecture was however of the ordinary type—fracture-dislocation from indirect violence,—and I felt that operation was not desirable, even though the presence of the deep reflexes showed that we had not a complete transverse crush to deal with.

The rapid return of sensibility when I first saw him was certainly an encouraging sign, but the lesion was so high up, and associated with a fracture of the sternum, that I could not give a good prognosis. But fortunately improvement was rapid, and I am able to record one of those rare cases of fracture-dislocation in the upper dorsal region with lesion of the spinal cord ending in recovery, for with the exception of the still exaggerated knee-jerks he has recovered.

The tendency to death in these cases is usually from paralysis of the intercostals. The diaphragm acts, and the patient can inspire, but cannot cough or make any forced expiration; hence the lungs become congested, and the bronchial tubes filled with mucus, a low form of basic pneumonia is set up, and death occurs in a week or two (often earlier) after the accident.

There is another method of treatment which might have been adopted in this case—reduction of the deformity of the spine under an anæsthetic. But most surgeons consider that the risk of further damaging the spinal cord by this method is so great that it is not to be recommended. Considerable force might be necessary to separate the vertebræ and get them into better position, and the movement might not be sufficiently under the control of the operator to prevent a further crush of the cord, for when bony points are firmly interlocked and then suddenly give way, they are very likely to nip the cord in their recoil.

Digitized by Google

MECHANICAL OR TRAUMATIC ARTHRITIS.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

(*Concluded from p.* 363.)

I WILL now describe the other conditions which result from the transmission of excessive force through a joint, or to its transmission in a specialised manner.

Owing to a want of familiarity with pressure changes, and consequently with the normal physiology of the skeleton, surgeons describe as the product of a disease the alterations in structure that we know to result from a physiology different from the usual, or what is called the *normal*, and which are perfectly characteristic of the anatomy of the individual in his peculiar mechanical relationship to his surroundings. This is accounted for by the fact that the presence of any deviation from the normal in the form of a bone or joint, such as partial or complete ankylosis of an amphiarthrodial joint, the conversion of an amphiarthrodial into an arthrodial type, the eburnation and limitation in the range of a freely movable joint, the development of a completely new one, etc., invariably suggest of necessity to their minds some antecedent inflammatory condition.

The chief blame for this want of knowledge rests with the anatomist, who either ignores altogether the consideration of the physiology of the skeleton, or describes it in the most unscientific manner possible, usually incorrectly.

As a matter of fact, it is quite impossible to arrive at a correct knowledge of the mechanics of the skeleton from a study of the normal body alone, and, to my knowledge, no anatomist has ever taken the trouble to analyse the several postures of activity or of rest which are afforded to him in abundance and in the greatest perfection by the skeletons of labourers and of feeble children.

He has been satisfied to leave anything abnormal in the joints to the surgeon, if he escapes the temptation to call it a congenital abnormality. The surgeon, on the other hand, has no difficulty whatever in dealing with such conditions of this sort that he does not understand, since he calls them osteo-arthritis or rheumatoid arthritis—terms which he applies indiscriminately.

To attempt to explain the several changes that take place in the skeleton from the exercise of force other than on a single occasion, it is necessary to formulate a few statements or general laws.

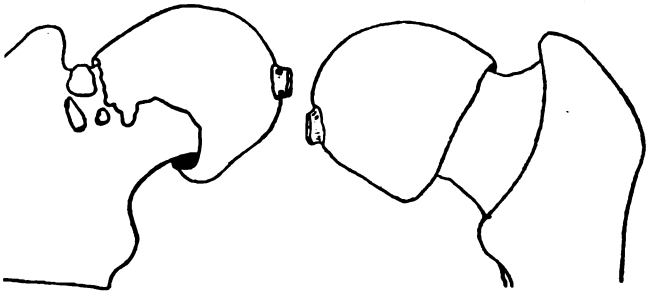
1. The skeleton remains normal only so long as it performs a certain combination of *movements of activity*, and assumes *attitudes of rest*. In other words, the individual must bear a definite mechanical relationship to his surroundings. This relationship varies in different races, and, in consequence, the average individual of each race has a normal anatomy of his own. Anatomists have attempted to obtain a general idea of this normal condition by a kind of composite photograph or series of measurements called indices, with results which are clearly far from satisfactory.

This does not appear to be the right way to study the causes of things, or to establish general principles.

2. The performance of the several physiological functions of feeble children is imperfect. They assume habitually attitudes of rest in proportion as they perform their respiratory function inadequately. These attitudes become fixed later, because the rate of bone formation in the several portions of a growing line varies inversely with the pressure transmitted through them. This produces an alteration in the relationship of the epiphysis to the body of the bone, and a change in the form of the ends where they come into contact with adjacent bones. Besides the necessary alteration which ensues in the shape of the articular surfaces, consequent on their altered mechanical relationship to one another, their area is increased by the formation of bone upon their edges. In the case of movable joints this marginal osseous formation becomes covered by articular cartilage. As time goes on, and the patient passes the meridian of life, the opposing surfaces of articular cartilage are removed at the points of greatest stress, and the layer of bone exposed by its removal is rendered dense and eburnated. Associated with this there is a progressive destruction of the opposing bony surfaces, with the formation of additional bone on their articular margins. The object of this is to render the joint more secure. At this period in the history of the individual this new bone is not covered by articular cartilage.

Digitized by Google

3. If a vigorous subject assumes habitually the same attitude or sequence of attitudes of activity, the posture adopted at the time becomes fixed, and later exaggerated. This results from a partial or complete removal of the soft parts between the bones. The extent to which this takes place depends on the amount of weight transmitted through the part. Associated with this there is abundant formation of bone on the margins of the articular surfaces, and in the case of the less movable joints these masses tend to unite to one another, so rendering the part more secure and capable of bearing more efficiently the great stress thrown upon it. Strain exerted upon muscles and ligaments tends to convert the former into tendon and the latter into bone, forming projections on bony



FIGS. 7 and 8.—Normal right femur of coal-trimmer.

surfaces, or ankylosing adjacent bones to one another. At the points of greatest stress the articular cartilage is removed, and the surface of bone which is exposed is rendered ivory-like in density. Besides the limitation which takes place in movable joints, there occasionally develops an increased mobility in others. In some cases a considerable displacement of bones upon one another may ensue, as in spondylolisthesis.

I have chosen a few specimens to demonstrate some of those changes. For instance, Figs. 7 and 8 represent the normal right femur of a coal-trimmer diagrammatically. They illustrate the remarkable alteration in the form of the head, whose area is considerably increased by bone formation, articular cartilage being formed upon its surface, producing a mushroom-like appearance. On the front of the neck is seen a nob of bone which impacts against the margin of the acetabulum in flexion of the hip-joint. I need hardly remind the reader that the anatomist labours under the delusion that flexion of the hip-joint is limited by the apposition of the thigh against the abdomen.

This is clearly absurd, since flexion of this joint is controlled by the impact of the neck of the femur against the margin of the acetabulum. The angle of possible flexion varies inversely with any adduction, and directly with any abduction. When combined with very moderate adduction the degree of possible flexion is very small indeed. This impact is a matter of great practical importance since it is upon the leverage action exerted through the femur, with the point of contact of its neck with the acetabular margin as a fulcrum, that fractures of the neck of the femur, separation of its epiphysis, and dislocation of the head of the bone depend.

The femur of the coal-trimmer shows exceedingly well the point of habitual impact, as represented by the prominent mass of bone, and by the adjacent groove.

The eburnation of the area of the head through which the greatest pressure is transmitted is not shown very clearly.

Fig. 9 illustrates the elbow-joint of the coal-trimmer looked at from its inner aspect. It is in extreme flexion. This, like the degree of extreme extension, is much less than the normal, the elbow-joint of this labourer only permitting of movement through a limited range. This limitation is very advantageous to the individual following this particular occupation. As this illustration shows, there is much new formation of bone upon the margins of the articular surfaces, by means of which the security of the joint is enormously increased.

Fig. 10 represents the anterior surface of the lower end of the humerus, and the buttresses of bone which have formed in the coronoid depression and about the point of impact of the margin of the head of the radius. The masses which come into contact with the apex of the coronoid process and with the head of the radius limit the flexion of the elbow-joint already alluded to. Similar changes exist in the olecranon fossa. The photograph indicates also the eburnation of the radial head through which the greatest part of the force is transmitted, the surface with which the ulna articulates being seen to be covered with articular cartilage that has fissured in the dry specimen.

Fig. 11 illustrates the upper end of the radius from the same labourer, and the changes this bone

Digitized by Google

undergoes. These are too clearly shown to need description.

Any alteration in the mode in which pressure is transmitted through a joint brought about by such a change in the mechanics as ensues when a bone is broken and its fragments are united with their axes, forming angles with one another, as is usually the case, is followed by very definite changes in the form and structure of the affected joints. These vary largely with the age of the patient, as in the conditions previously described. For instance, in the young and vigorous subject bone and articular cartilage are formed in great abundance, so enabling the joint to perform its altered function more or less securely and efficiently after a certain time has elapsed. If, however, the subject of the injury is no longer young and vigorous (this period being reached at a much earlier age in the labourer than it is in those leading a less arduous life), the articular cartilage is removed at the points of greatest stress, the subjacent bone being exposed, and undergoing sclerosing and frictional changes. The articular area is increased by a more or less abundant formation of new bone (popularly described as "lipping"), by means of which the security of the joint is increased. Articular cartilage is no longer formed in advanced life, and the tendency is towards destruction of opposite surfaces rather than towards new formation. This is very much more marked should there co-exist any im-

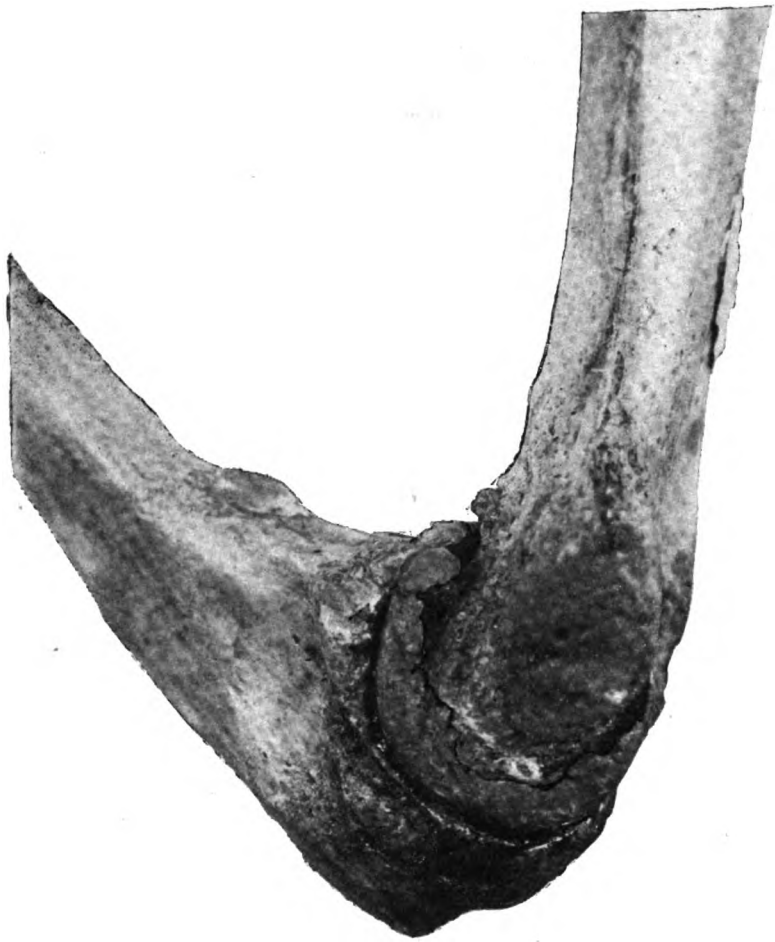


FIG. 9 illustrates the elbow-joint of the coal-trimmer seen from its inner aspect in a position of complete flexion. (From *The Practitioner*, by permission.)

Digitized by Google

pairment of sensation in the affected part, such as one sees in the ataxic condition or in old age.

Fig. 12 represents a vertical median section through the cervical spine of a labourer. It shows very well the destruction of the soft parts between several of the bodies of the vertebræ resulting in complete ankylosis of the fifth and sixth, with ossification of the ligamenta subflava, under the influence of excessive pressure in the one case and excessive strain in the other. The bodies of some of the other vertebræ are also altered in form.

Fig. 13 shows the fourth to the ninth dorsal

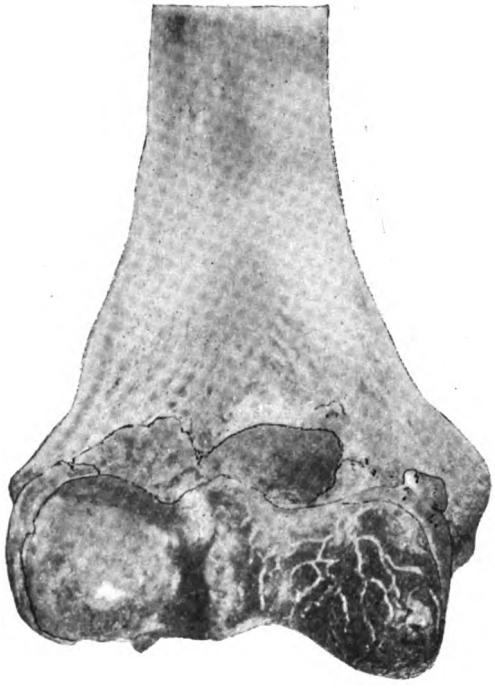


FIG. 10 represents the anterior surface of the lower extremity of the humerus of the coal-trimmer. (From *The Practitioner*, by permission.)

vertebræ in vertical median section. This was obtained from the body of a labourer. The function performed by the dense supporting callus in enabling the spine to support its load is so obvious in the drawing that I need not explain it further.

Figs. 14 and 15 represent vertical transverse sections through the acetabular cavities of a charwoman whose anatomy I described in the 'Guy's Hospital Reports,' 1886-1887 :—"Some changes in the form of the acetabular cavities, spinal column, and joints of the hand, illustrated by the anatomy of the charwoman."

In Fig. 14 the bony and cartilaginous articular surfaces have been extended downwards, so as to encroach to a very considerable extent upon the area of the original non-articular cavity in the floor of the acetabulum.

FIG. 11 represents the upper extremity of the radius of the coal-trimmer. (From *The Practitioner*, by permission.)

In Fig. 15 the non-articular area is normal in form and extent. The upper portion of the cavity

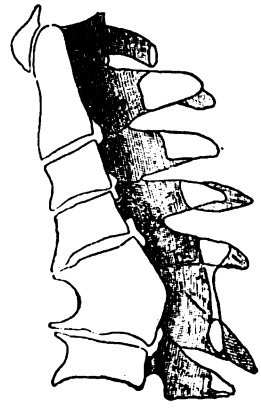


FIG. 12.

has been extended outwards by a deposit on its margin, while the upper concave aspect of the

Digitized by Google

acetabulum is deprived of its articular cartilage the exposed bone being somewhat eburnated.

4. That pressure exerted directly upon bones alters their form in a manner varying with the

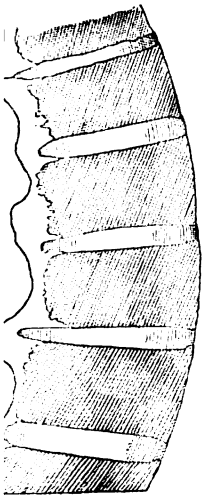


FIG. 13.

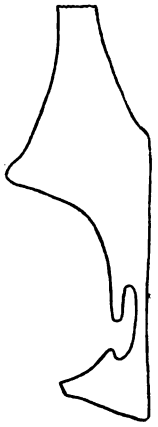


FIG. 14.

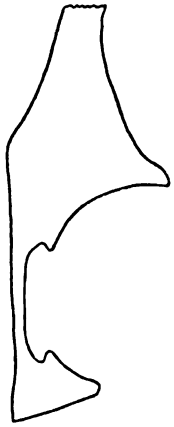


FIG. 15.

character of the pressure is shown by the sternum of the shoemaker, and the progressive section of the lamina of the fifth lumbar vertebra in the spondylolisthetic condition of the coal-heaver, and by the divided fourth lumbar lamina in the fully developed coal-trimmer, etc. (Figs. 16, 17, 18).

FIG. 16 represents the fourth and fifth lumbar vertebræ and the sacrum of the coal-heaver in vertical antero-posterior section, and illustrates the form of spondylolisthesis found in this class of labourer.

5. That excessive movement readily alters a joint from the amphiarthroidal to the arthroidal type. This is shown exceedingly well by the arthroidal joint which develops between the bodies of the lumbar vertebræ in the coal-trimmer, the developing joint in the ossifying cartilage of the first rib, etc. These are shown in Figs. 17 and 19.

Precisely similar changes occur in joints whose mechanics have been altered by the fixation of another articulation in any position. For instance, in ankylosis of the hip-joint in a position of flexion

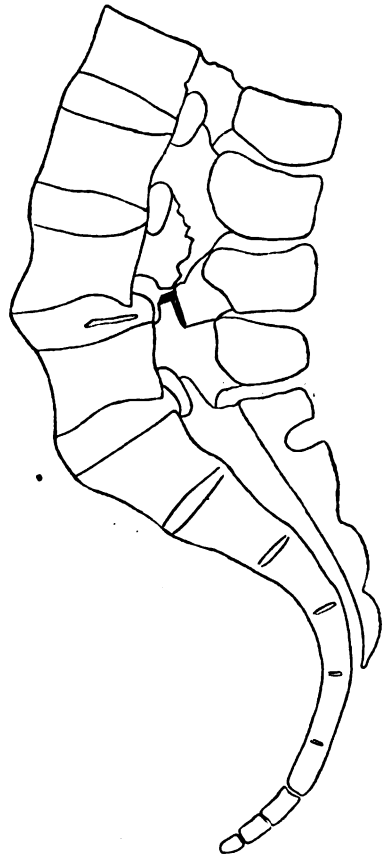


FIG. 17 shows a vertical median section of the lower part of the spinal column of the coal-trimmer. The superior articular process of the fifth lumbar vertebra is flattened superiorly where it articulates with a corresponding surface on the inferior articular process of the third lumbar. The cavity in the fibro-cartilage between the bodies of the fourth and fifth lumbar vertebræ is also represented. The articular processes of the third and fifth vertebræ have gradually cut through the intervening portion of the lamina of the fourth during the over-extension of the spine and the succeeding rotation, so that there is first a violent impact of the processes into the lamina followed by a lateral grinding movement. Finally the two articular processes come into contact and change results in consequence.

and adduction the mode in which pressure is transmitted through almost all the joints in the trunk and legs is changed, and in every one of

Digitized by Google

these definite anatomical conditions develop in a proportion varying with the degree in which their physiology is altered.

Such a variation in the physiology of a joint or joints may be brought about by external pressure, clearly dependent upon an alteration in the function or in the physiology of the part. How well this condition shows the difficulty the surgeon experiences in avoiding the mistake of confusing the cause with the effect! When they are forced

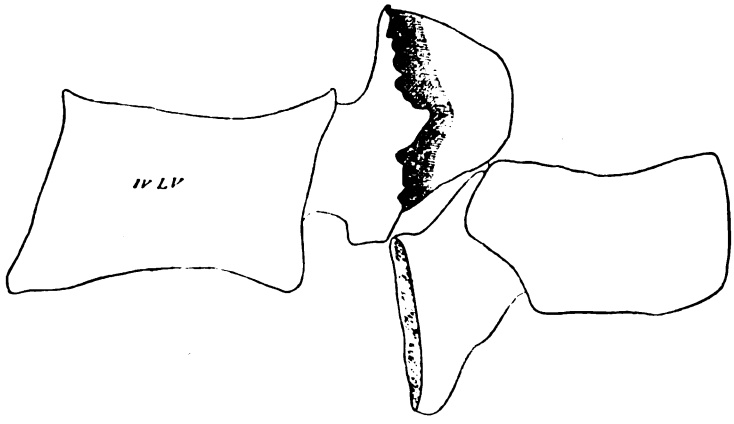


FIG. 18 shows the separate portions of the fourth lumbar vertebra in their relative position. This condition has been observed by several, and has been regarded as resulting from congenital non-union of the bony centres.

as in displacement outwards of the great toe by the pressure of the boot exerted on its inner aspect, or

FIG. 19 shows the form of spondylolisthesis that occurs in such labourers as carry heavy loads on their backs less heavy than those borne by the coal-heaver, and who pick them off and replace them on the ground. The arthrodial joint seen between the fifth lumbar vertebra and altered upper piece of the sacrum is produced by this greater freedom of movement.

on its extremity, or on both. I have illustrated in Figs. 20, 21, 22, 23, and 24 these so-called osteo-arthritic changes, which are regarded by many as being the cause of the deformity, but which are

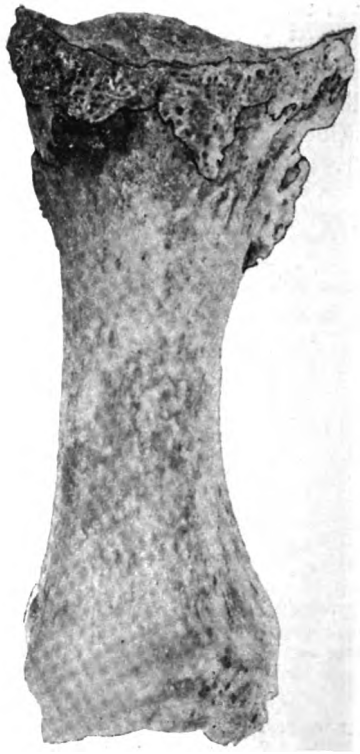


FIG. 20 represents the upper surface of the first metatarsal bone of a labourer who was engaged in carrying heavy loads on his back. (From *The Practitioner*, by permission.)

Digitized by Google

upon his attention, he notices that these mechanical changes coexist in a more or less marked manner with the several deformities of the toes, and he at once regards them as factors in their causation.

Fig 20 represents the upper surfaces of the first metatarsal bone of a labourer, whose great toe was fixed in a position of very slight extension upon the metatarsal bone, the deviation actually being but slight. He had always worn boots too short for him, and excessive pressure had been exerted upon the opposing surfaces of the phalanx and metatarsal bone, partly by the impact of the extremity of the toe against the boot, and to a much greater degree by the strain exerted upon the toe by the extensor and flexor tendons when a heavy load was being carried. This produced the alteration in the form of the head shown in the illustration, the eburnation of its flattened extremity, and the heaping up of irregular masses of bone around the extended articular surface. All these changes must arise in consequence of the variation in the physiology of the part, and can have nothing whatever to do with any imaginary disease. They occur in obedience to the most simple mechanical laws. Owing to the excessive force transmitted through the head of this bone to the subjacent sesamoid bones, the opposing surfaces are ebur-

FIG. 21 represents the upper surface of the first metatarsal bone and its phalanges obtained from a female subject. It shows the changes that ensue normally in the foot of the woman from wearing boots. (From *The Practitioner*, by permission.)

FIG. 22 represents the first metatarsal bone and phalanges obtained from the body of a labouring man. It shows the several changes that result partly from boots and partly from a very laborious occupation. (From *The Practitioner*, by permission.)

Digitized by Google

nated, and their areas enormously increased by osseous marginal lipping.

FIG. 23 represents the first metacarpal bone and phalanges of the shoemaker seen from the side. (From *The Practitioner*, by permission.)

the latter in a powerful labourer. In the woman's great toe, note the atrophy of the inner portion of the head of the metatarsal bone which follows upon its loss of function, and the increase in size and the alteration in the form of the outer part.

In the case of the man there is, in addition, a much more abundant marginal deposit upon the opposing surfaces of the phalanx and metatarsal bone.

In both cases the terminal phalanx shows the asymmetry which results from pressure exerted upon it laterally.

Fig. 23 shows the lateral aspect of the normal thumb of an old shoemaker whose bones are retained in the relationship which they bear to one another when the thread is grasped and strain exerted upon it. The excavation and the eburnation of the base of the metacarpal bone cannot be recognised very clearly in the photograph, but all the results of the transmission of great pressure are illustrated very well indeed in the metacarpo-phalangeal and interphalangeal joints as eburnation of opposing surfaces, and as alteration in their shape and extent by abundant formation of new bone.

Fig. 24 is an excellent example of the change which a bone undergoes normally in old age, because of an alteration in the mechanics involved in the altered physiology of the part. The glenoid cavity with which this articulated was quite flat,

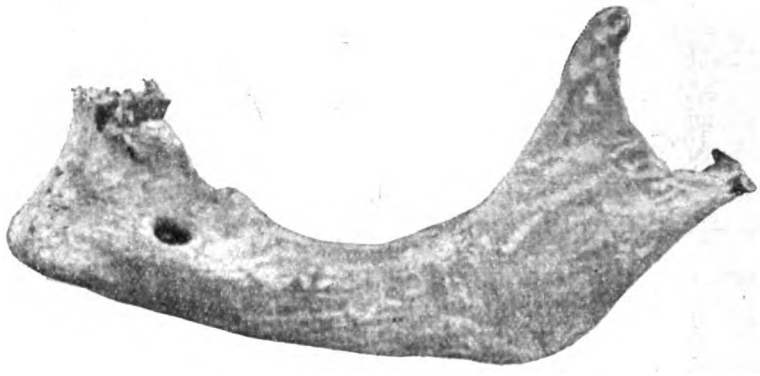


FIG. 24. (From *The Practitioner*, by permission.)

Figs. 21 and 22 represent the upper surface of well-marked examples of displacement outwards of the great toe, the former being in a woman, and the eminentia articularis having been completely rubbed down. There was no vestige of a fibro-cartilage.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 467. **WEDNESDAY, OCTOBER 9, 1901.** **Vol. XVIII. No. 25.**

CONTENTS.

PAGE

*With Dr. Lees at the Hospital for Sick Children 385

*A Demonstration of Cases of Skin Diseases. By PHINEAS S. ABRAHAM, M.D., F.R.C.S.I. 392

*A Clinical Lecture on Cardiac Pain and its Treatment. By ARTHUR LATHAM, M.A., M.B.Oxon., M.R.C.P. ... 396

**Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

WITH DR. LEES AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

May 16th, 1901.

LADIES AND GENTLEMEN,—Here is the youngest case of acute nephritis which has ever come within my observation. The patient was only two years and four months old when he was admitted on October 26th, 1900. He was then markedly dropsical, and his urine when boiled was solid with albumin. The child had always been healthy till two months before, when he had an attack of diarrhœa lasting three weeks. He recovered from this, and seemed well until about two weeks before, when he was observed to be "growing fat"; but it was soon found that this fatness was partly due to œdema. His urine was scanty and high-coloured. There is no history of scarlet fever or of sore throat. There had been no desquamation, and none was observed on his admission or since. His mother had suffered from dropsy three weeks before the child was born and for some time afterwards. Her father and brother are said to have died of Bright's disease, so that we may suspect that there is a tendency to renal disease inherent in the family.

On admission he was a fat child with pale face, a temperature of 98·6°, a pulse of 128, and a respiration rate of 34. There was œdema of the feet, and marked puffiness about the eyes, fingers, and hands; the umbilicus was projecting; there was dulness in the flanks, which gave place to resonance when the child was turned on one side. The edge of the liver was two fingerbreadths below the costal margin. The spleen was not palpable. In the lungs were numerous moist râles over both sides, back, and front, due no doubt to bronchitis and œdema of the lungs. The urine contained so much albumin that it was almost solidified on boiling.

On November 2nd the note is, "Œdema of the feet and legs slightly less. Still some ascites. In the urine the albumin is slightly less, but still

Digitized by Google

very abundant. In the lungs considerable bronchitis."

On November 3rd the œdema was much less; in the urine the albumin was less abundant; microscopically there were seen to be hyaline and granular casts, red corpuscles, and epithelial cells.

On November 5th the œdema is noted as having almost disappeared, except from the feet; but there is still some ascites. The albumin in the urine is much more abundant than on the 3rd inst. The bronchitis is improved, but there are some snoring râles over both lungs. In the heart there is accentuation of the second sound at the apex and in the aortic area,—the increase in tension in the arterial system which one finds ordinarily in acute and chronic nephritis.

On the 6th the note is, "Albumin in the urine still very abundant; œdema and ascites practically gone. Still some bronchitis."

So that at first in the hospital he showed a considerable tendency to improve, and that improvement seemed to be largely due to the use of constantly repeated hot packs, in which the child perspired freely. I will quote a few further extracts from the notes.

November 20th, "He was worse; œdema of the feet more pronounced; also œdema of the face; no ascites now; dulness at the base of both lungs with scattered sonorous rhonchi; skin dry; slight cough; a patch of erythema on the forehead."

November 28th: "Œdema more marked than it was some days ago. Urine: albumin very abundant. Bronchitis slight; skin dry."

December 7th: "Blotchy papular erythema on the cheeks, nose, and forehead."

December 13th: "He was again still worse, and had vomited. There was a great diminution in the quantity of urine passed. Temperature this morning, 101·5°—no cause ascertained. Urine alkaline, loaded with albumin."

December 14th: "Perspired very little, but did not vomit. Temperature 98°."

December 17th: "Dropsy increasing again, especially about the face."

December 31st: "Marked dropsy of face, hands, and feet. The patient very drowsy; does not perspire at all in his wet packs. No cough; very small quantity of urine passed."

January 4th: "Dropsy very much increasing. Some cough. Pilocarpine injections used, beginning with one twelfth of a grain, and now increased to one eighth of a grain. He perspired yesterday well after the injection. The temperature yesterday morning was 102·2°, and 97·8° the previous evening. Urine loaded with albumin."

January 7th: "Temperature raised, fauces red and injected; no exudation."

January 15th: "Condition worse, much œdema, patient drowsy and irritable if disturbed. Vomits occasionally; much albumin."

At this date, you see, it was about as hopeless a picture of Bright's disease as you could imagine.

January 24th: "Temperature rose last night to 104·2°, nothing in the throat except some injection and enlargement of the tonsils."

January 28th: "Erysipelatoid swelling of the subcutaneous tissue round right eye, with red advancing edge."

February 5th: "Legs punctured for drainage; Southey's tubes were placed in the dorsum of the feet, and drained fairly satisfactorily."

February 7th: "Erysipelatous appearance round punctures of the left foot; fomentations applied."

February 11th: "Legs punctured again."

February 21st: "Suppuration in both legs around punctures; inflammation round the left eye, and upper lip, and right ear; œdema and coldness of the extremities."

February 26th: "Improvement began, inflammation round left eye subsided, but a new patch developed round the angle of the mouth on the right side. Right ear improved."

February 28th: "Left leg improved; discharge almost ceased; right leg discharging a good deal."

March 5th: "Swelling of hands much less than formerly, coldness of extremities not quite so marked. The child can now open both eyes. Redness round the right ear gone; the condition round the angle of the mouth much improved."

March 16th: "The œdema gradually increased till the right eye is almost closed again."

April 8th: "Child much better; legs punctured for the third time on the 6th inst."

April 26th: "Urine contains phosphates, but comparatively little albumin."

May 2nd: "Urine 1012, slightly alkaline, albumin great in quantity."

May 9th: "Urine 1020, slightly alkaline; still contains much albumin."

The child is still somewhat drowsy and irritable,

Digitized by Google

but he certainly is a good deal improved from what he was a month ago, when I really thought nothing more could be done for him. Various means of treatment have been used. For a long time he had a hot pack every evening to make him perspire, and when that failed we tried the effect of subcutaneous injections of pilocarpine, which certainly had some success. After a time we were compelled to draw the fluid off from his legs, because they were so much swollen, and we endeavoured to assist the circulation by giving him digitalis. That had no very marked result, but when we added caffeine it produced some definite effect; at any rate, the improvement seemed to begin at that time, though whether it was in consequence of the caffeine being added I do not know. Probably some part of the improvement which has resulted is to be attributed to the development of a considerable amount of diarrhœa. No doubt loose motions tend to relieve the distressed kidneys, though they have disadvantages in other respects. I thought you would like to see the case as one of typical acute nephritis at an extremely early age, and with a prognosis which, in spite of the present improvement, must be considered as of the gravest possible order. I do not see how it is possible for a nephritis of so great severity, at such an early age, to disappear.

There is an interesting question about the causation of an attack of nephritis like this. I daresay you all feel that the ætiology of acute nephritis is in a very unsatisfactory condition. We know that acute nephritis may be a result of scarlet fever; that is certain. And then the text-books tell us that exposure to cold and wet may produce acute nephritis. Those two were for a long time the only recognised causes of acute nephritis, except diphtheria. But, as in other departments of medicine, you know that "cold and wet" as a cause of disease has had to give place to a bacterial ætiology, and to take a secondary position in accounting for the symptoms. For instance, pneumonia, acute rheumatism, pericarditis, peritonitis, and some spinal cord diseases, as myelitis and poliomyelitis, were formerly ascribed to cold and wet. But one sees now that in many of these diseases, although the chill resulting from the exposure to cold and wet favours the action of various organisms, it is in reality the organisms which are mainly responsible. In pneumonia we all know that the disease is due to the development in the lungs of a definite organism, and yet exposure to cold and wet is often a contributing factor. I remember some years ago some experiments by a German observer, who made various animals inhale air which was loaded with the pneumococcus. But he did not produce pneumonia by that means until the animals were exposed to cold; then they developed the disease. That shows that under ordinary circumstances they were able to resist it. So that what was formerly looked upon as the cause of pneumonia is now found to be only a favouring condition, and not the true cause. Similarly with regard to pericarditis. That may, perhaps, come on after cold and wet, but it is due usually to the rheumatic process, and it is now nearly certain that rheumatism is due to an organism. Similarly, again, with regard to peritonitis; we think less of cold and wet now that we have learned the results which follow from disease of that troublesome anatomical arrangement—the appendix vermiformis. Similarly with regard to diseases of the cord; it has become more and more probable that myelitis, and perhaps also acute anterior poliomyelitis, is due to organisms, though of that there is still no certainty. And so when one comes to inflammation of the kidney, one sees that though cold and wet may be a real factor, this will not explain the whole condition, and therefore we believe that in many cases there is a definite micro-organismal process which is responsible for the inflammation. Of that we know but little as yet. Still it may be said with practical certainty that scarlet fever must be a bacterial disease, although the organism is not known. Certainly diphtheria, of which the organism is well known, tends to affect the kidney by its toxins, and may, perhaps, cause nephritis. We find influenza occasionally affecting the kidney; occasionally also in typhoid fever there is a very definite nephritis present.

What is the cause of acute nephritis like this in a child two years old, in whom apparently scarlet fever may be excluded? One is at a loss to say what it is. But it seems to me that the fact of the child having previously had an attack of diarrhœa lasting three weeks suggests that he may have had microbic poisoning of some kind—some organismal process in his alimentary canal

Digitized by Google

which may have produced an infection of the system, and so a secondary inflammation of the kidney. That seems possible. The causation of acute nephritis is a subject which requires much more investigation.

It seems not impossible that rheumatism itself may be one of the causes of nephritis. If rheumatism is an organismal disease, it is possible that, as in diphtheria, the elimination of its toxins may cause disease of the kidney, or that, as in typhoid fever, the organisms themselves may pass through the kidney into the urine. A rheumatic ætiology is suggested by the history of another patient suffering from renal disease.

Here is a little girl of eight and a half years of age, who was well until six months ago; then she began to feel tired. Four months ago there was a swelling of the eyelids and ankles. She has complained of headache for the last day or two. She had been for four months under the care of a doctor, who said she was suffering from inflammation of the kidney. The urine has been "thick;" no abnormality in frequency or quantity has been noticed. She had measles twelve months ago. She has had whooping-cough, but has never had scarlet fever; neither has she had rheumatism, or sore throat, or chorea. This girl's mother suffers from rheumatism, and four of the mother's seven brothers and sisters have had rheumatism. The three others all suffered from dropsy. This history suggests the possibility that in the different members of this family the same poison may have produced a different effect, according to the different resistance of the various tissues in the several individuals. In this child the renal disease developed quite slowly and insidiously; here was not at any time an acute attack of dropsy, as in the case of the little boy I have just shown you. Apparently there was not at any time blood in the urine. The disease came on gradually, with only a little œdema and a little headache, and with practically nothing else. But when she came into the hospital it was found that the urine contained albumin in considerable quantity. It was alkaline, with a specific gravity of 1015, and, on boiling, a copious deposit of albumin; no blood, no sugar, no casts. But this rather scanty indication of nephritis was accompanied by a condition of heart which seemed to show that the condition of the kidney was really one of some standing, for her heart was large, and it had sounds which seemed to justify the inference that the enlargement was not due to rheumatism, but was due to the high tension of renal disease. For the heart extended more than two finger-breadths to the left of the left nipple line, and the second sound at the apex and over the base of the aorta was accentuated. The first sound was somewhat short, and the first cardiac interval between the first sound and the second sound was longer than normal, showing that there was a considerable rise of tension in the arterial system, and that that rise of tension had told upon the heart, and had caused this chronic enlargement. Her eyes were examined, but nothing was found amiss with her optic discs or with the retina.

In some cases of chronic renal disease in small children we have had evidences of the same form of albuminuric retinitis as is found in older people, that is to say, white spots in the retina and hæmorrhages; sometimes a little optic neuritis. There was nothing of that in this patient.

The last note about the urine is May 10th: Improved in specific gravity (1018), acid, a cloud of albumin. It was centrifugalised, but no casts have been found.

Thus we have a patient with an insidious form of renal disease, never intense, slowly developing, and yet producing apparently a considerable effect upon her heart, showing that there must be more disease than one would infer at first from the symptoms and the condition of the urine. Here, again, in the absence of any indication of scarlet fever, what is the cause of chronic renal disease beginning in a small child without any obvious indication of cause? One falls back upon that curious statement in her family history, that some of the family have had rheumatism, others have had dropsy.

I should like to point out the difference between what one takes to be a "renal heart" and the more common forms of chronic heart disease,—which we have here in considerable numbers—from rheumatism. Both hearts may be pretty much the same size, but as a rule in the rheumatic hearts you find very obvious murmurs. There is almost always a systolic murmur, and sometimes a presystolic murmur also.

By way of contrast with this "renal" heart in which there is no murmur to be heard, I show you

Digitized by Google

a rheumatic heart in examining which you will feel very plainly the presystolic thrill of a contracted mitral. The heart of this child is about the same size as that of the child with renal disease, but you will be able to feel in this one a presystolic thrill, and if you listen you will hear a characteristic presystolic murmur and a short sharp first sound.

It is not very common to find evidence of advanced mitral stenosis in such young subjects. Every now and then we have such a case, in which the evidences of mitral stenosis are pronounced,—a long, rough presystolic murmur of the churning quality which one meets with in adolescents, and a thrill which can be felt, also of presystolic time. The mitral stenosis which one meets with in young children is usually much less advanced. I take it that mitral stenosis is almost always of rheumatic origin in early life. It is certainly never congenital, and if you watch it in the early stages you will see that it begins usually with a blowing systolic murmur, to which is soon added a double second sound, and shortly afterwards, perhaps even earlier, you may notice a very short murmur of presystolic time. I say of presystolic time in order to point out to you that the murmur at that early stage is usually of a blowing character, and not of a coarse vibratile character, such as we usually associate with mitral stenosis. As time goes on this blowing presystolic murmur becomes longer and more vibratile, until it gradually, as the orifice contracts, assumes the churning character which we know in young adults, and which in rarer cases you find even in young children.

I remember one post-mortem here on a child of nine, in which the mitral stenosis was very advanced; but this is unusual. You generally find that the valve is thickened and opaque, and slightly contracted; but to find a large amount of stenosis, so that the orifice admits only one finger, is comparatively rare. The reason is that the process is a chronic one, and takes some years for its complete development, so that a presystolic murmur in a child will often be found to be associated post mortem with only a slight amount of stenosis of the orifice; but usually there is some thickening and opacity of the valve segments.

The autopsy to which I am referring was interesting in this respect, also, that the pulmonary artery was atheromatous, whereas the aorta was not, showing the effect of the long-continued strain on the walls of the former vessel.

Pleural Effusion.

Here is a child who was admitted a week ago with a serous pleural effusion. The right side was normal, but the left front was dull. The heart was displaced, the breath-sounds weakened. Behind there was a dull note at the base extending to the angle of the scapula, and in the axilla to the sixth rib. Between the spine and the scapula there was bronchial breathing. Puncture proved that there was serous fluid in the pleural cavity. There are two points I want to make with regard to the case, one concerns the question of treatment, and the other is as to the real nature of the disease.

In such cases I aspirate at once, and draw off as much of the fluid as will easily come, and then compress the chest on that side by means of bands of strapping, to supply for a short time the place of the fluid pressure. Then over the strapping I put one or two large ice-bags. The effect of the cold is, I think, to check considerably the tendency to re-accumulation of fluid. This child had ice-bags on for two days, and when they were removed at the end of that period it was found that there was practically no re-accumulation. The ice-bags have been off for some time now. There is a little dulness remaining at the base, but even there it is not absolutely dull, and in the axillary region the resonance is very fair indeed. You can hear that there is a rubbing going on between the pleural surface of the lung and the pleural surface of the thoracic wall, showing that the fluid has not re-accumulated, and the union—which is a necessary part of the cure—is tending to be produced. I ask you to note, as proof that there has been no re-accumulation, that the surfaces are now rubbing, whereas before they were separated by fluid.

What is the real nature of the serous effusion into the pleura? Suspicion has been growing of late years that the essential nature of a large number of such cases is tuberculous. Many such cases of what we used to consider a comparatively innocent affection afterwards develop signs of phthisis. The matter was investigated statistically, and it was found that a large proportion of them became phthisical. But I want to point out that if you practise your percussion carefully you can

Digitized by Google

often demonstrate in the patient himself that there is evidence already of tuberculous consolidation. I have been struck with that in many cases of serous effusion. Sometimes in the lung on the side from which the fluid has been drawn off, sometimes in the opposite lung, you will find there is tuberculous disease.

This boy has dulness at the base, a thickened pleura, and possibly a little fluid remaining. As we ascend, the lung is quite resonant until we come to the posterior part of the apex, where it is dull. In front it is resonant until we come to a suspicious place close to the sternum in the first space, and there it is decidedly dull. If you will practise that for yourselves in cases of serous effusion, you will find that even when one side is full of fluid, and you cannot therefore be sure about the apex of that lung being tubercular, you will often be able to detect a focus of tubercular consolidation at the upper or lower apex of the other lung.

Neglected empyema.

Here is a little girl æt. 2. She was admitted with empyema of her left side. I want to draw your attention to this because of the statement that she had been quite well ten weeks before admission; that then she had "congestion of the lungs," and was attended by a doctor for five weeks, but got no better, and was brought here because of a swelling over the ribs. One cannot always accept the statements of patients; but, if it is true, one wants to know how it was that the doctor who attended her did not discover that there was a dulness over the base of the lung. Why was it that the child was allowed to go on for ten weeks, until the pus, despairing of being let out by surgical interference, tried to let itself out by making a spontaneous opening in the front of the chest? The child came in with the side dull all over, and a swelling in the position I show you. An opening was therefore made behind at once, a piece of rib was removed, and a drainage-tube put in. Some ten days afterwards, as the anterior swelling did not subside, a small incision was made over it, and a small abscess was found there; that was drained locally. You can hardly imagine the improvement in the appearance of the child compared with her condition on admission. She was then wasted and miserable, and her chest was much distorted. The removal of the pus, and the careful nursing she received in the hospital, have resulted in a most remarkable improvement. The empyema has been going on so long, that one cannot expect it to recover very rapidly.

At first there was no indication of any expansion of the lung, but now I am glad to say that the breath-sounds can be heard halfway down the chest; and although the empyema is still draining, yet there is an enormous improvement in the general shape of the chest, and one hopes that she may entirely recover. But if so, no thanks will be due to the doctor who allowed her to remain ten weeks without proper treatment, if that statement be true.

I think it may very well be true; for I remember some years ago a child sent into this hospital with a letter from the doctor, which I saw myself, and which stated that the child "had had bronchitis, and was sent to the hospital for change of air." You would hardly believe it, but it is a fact. This unfortunate child had one pleura absolutely full of pus, and died in a week from purulent meningitis. Such a performance as that on the part of the doctor is either gross ignorance or gross carelessness, and although one ought to be very charitable to a medical man in the strain and stress of busy work, one cannot help thinking that many men are far too careless in the investigation of their cases. It surely ought to be known by this time that pneumonia in children is often followed by empyema, and that in every case of inflammation of the lungs the greatest care should be taken to make sure that the dulness clears up, and that there are no remains of pus in the pleura.

Acute Chorea.

In regard to this next case I doubt whether any of you would suspect that he has been suffering from chorea. The patient lies there apparently fairly comfortable; but if you watch carefully you may see that under the clothes the left hand is twitching, though his face is at rest. But when admitted ten days ago the patient was in a condition of acute chorea, with extreme restlessness, and almost uninterrupted choreic movements.

Three weeks ago he became sleepy, did not eat well, and complained of pain and stiffness in the

Digitized by Google

knee-joint and pain in the legs. That means rheumatism.

On the 13th April the head and limbs began to twitch and the stiffness in the limbs disappeared, but he could not eat or stand. There was no vomiting, and no sore throat. The patient was drowsy during the day, and did not sleep at night. He had had very little sleep for three nights before admission. When admitted, he was pale and thin, and looked extremely ill; but now, ten days after his admission, I doubt whether all of you would be able immediately to diagnose it as a case of chorea. Therefore I think the question of treatment is of some interest, for certainly this child has recovered remarkably rapidly; and I have seen several other cases recover rapidly under treatment of the same kind.

What is one to do with these acute choreas? What is acute chorea? What is the meaning of the process? We know that that has been a debated subject for a long time, and we know also that, to a considerable extent through observations made at this hospital, the rheumatic nature of chorea is becoming more and more recognised. We know that the gross anatomy of chorea is summed up by such statements as hyperæmia, exudation, and in some cases local thrombosis or even embolisms. And it seems likely that what is really happening is that the brain is itself affected locally by the rheumatic organisms, and at the same time poisoned generally by the toxins they secrete. I show you now drawings by Dr. Poynton and Dr. Paine ('Lancet' May 4th, 1901) representing diplococci in the pia mater and in the epithelial cells of the cerebral capillaries.

Those drawings are not from the human subject, but from a rabbit in which choreiform symptoms had been produced by intra-venous injection. But the fluid which produced that result in the rabbit was pericardial fluid taken from a case of rheumatism in a child. So it is fairly comparable to human chorea, and I consider that observation as the first step in an investigation which will probably demonstrate to us that in chorea there is actually a rheumatic organism in the tissue of the brain itself, not only in the motor cortex but widely distributed in the brain.

Now if that idea is prominent in our minds, we shall see clearly that if the process is essentially rheumatic it is important to treat it on anti-rheumatic lines, and to treat it early; and we should expect that anti-rheumatic treatment, administered in a sufficient dose and sufficiently early, might tend to arrest chorea before it became constant. If the process is allowed to go on, if the organisms are allowed to elaborate their toxins and poison the cortical cells in the brain, and perhaps to produce actual degeneration of the nerve cells, and actual fibrosis of the cortex, you cannot expect the symptoms of chorea to subside rapidly under any treatment whatever. But supposing that that is the real meaning of chorea, an invasion of cerebral blood-vessels by the rheumatic diplococcus, then it is possible that by sufficiently energetic treatment at an early period you may be able to arrest the phenomena of chorea. Acting on that belief, we gave to this child when he came in seven grains of salicylate of soda with fourteen grains of bicarbonate every three hours. His age is seven—a grain for each year of his age. In addition, we endeavoured to allay the nervous irritation and to promote sleep by giving him bromide and chloral at night—five grains of chloral hydrate and six grains of bromide. During the first night two such doses were given.

In addition, finding that his heart was somewhat enlarged, and that there was a systolic murmur at the apex, and therefore evidence of rheumatic implication of the heart itself, I directed that an ice-bag should be applied over the heart. Those measures were persevered in, and there was remarkable improvement, as you see. The child has practically lost his chorea, and his whole look and appearance are totally different. He is now full in the face and bright, and has altogether lost the look of extreme illness which he had on admission.

The salicylate of soda is being still continued, and will be for some time. That is not a solitary case, or I would not have shown it to you. One ought not to advocate a plan of treatment founded upon a single case. But I have seen several such cases, in which vigorous anti-rheumatic treatment, employed at a sufficiently early stage of the chorea, has resulted in the symptoms rapidly subsiding. Therefore I think it is a treatment which is worthy of trial in cases in which the symptoms are recent. If the symptoms have been of longer standing the results of this treatment may be slight. That cannot be wondered at. It is important to get your choreas early and to treat them vigorously at once with anti-rheumatic remedies.

Digitized by Google

DEMONSTRATION OF CASES OF SKIN DISEASES AT THE WEST LONDON HOSPITAL.

By PHINEAS S. ABRAHAM, M.D., F.R.C.S.I.,
Dermatologist to the Hospital, and Surgeon to the Hospital for Diseases of the Skin, Blackfriars.

1. This young woman came to the hospital on Tuesday last complaining of an eruption which itched very much. When I first saw the patient I observed a number of glistening papules of a dark colour, which reminded one very much of lichen planus. On other parts, for instance on her arms, there were maculæ, especially on the flexor surfaces, and she gave a history of having had a very extensive eruption before. On further examination I found that there were enlarged glands in various parts of the body, and ultimately by careful questioning I obtained a history of a primary sore. At first the condition was difficult to distinguish from lichen planus, although it felt rough like lichen planus acuminatus. But there is nothing indicative of lichen planus about the lesions on the back. As a matter of fact the eruption is polymorphic. The enlarged post-occipital glands are very characteristic in this connection. She had a sore throat, which is rather better than it was. I have put her upon Hyd. cum Cretâ, gr. ij., with opium, three times a day, and she is using an antiseptic mouth-wash. It is an interesting case, as showing that occasionally the eruption of secondary syphilis is pruritic.

2. This woman presents a lesion which has only recently appeared. She complained of pain in the upper part of the brow, and the history is that there were little blisters which formed this mass. It is a case of herpes zoster. The lesions being broken and massed altogether, one could easily mistake it for something else, notably eczema, for which I at first treated it. I am giving her quinine and a protective application, and under that she will soon be all right.

3. This boy has the disease which is so very common in this part of London, namely impetigo contagiosa. Two or three days ago his face was very much crusted with the condition, but now it is very much improved. It is very easily cured by the application of Ung. Hyd. Ammon. of the pharmacopœia. I always instruct the mother to soften the crusts with warm water first. I do not believe in poultices, as they tend to promote, by their warmth and moisture, the growth of the pathogenic germs.

4. This girl presents a lesion, apparently of tubercular nature, which she has had for twelve months. It has always been in the same spot. Although in such growths the bacilli are rarely demonstrable by the microscope, their presence can be proved by inoculation experiments. It is really a form of lupus verrucosus; I do not like the use of the term "scrofulodermia" in this connection, because it should be reserved for cases of extension from tuberculous glands. The patient is at present using iodide of lead ointment, but I shall probably scrape the growth freely and apply acid nitrate of mercury.

5. This boy presents extensive psoriasis lesions on his face. There is a thick crust on the patches almost like a limpet-shell. He had psoriasis for a long time, and then became clear. After that his father sent him into the country, and he developed the condition again acutely. It is no longer to be believed that psoriasis must necessarily be confined to certain places; it may be seen on the abdomen, around the umbilicus, on the chest, arms, and in some of the cases it is not on the extensor surfaces at all, though it must be admitted, most of the cases have the extensor parts involved.

6. This young woman is the subject of ordinary urticaria. When she was here the other day it itched very much. She attributes it to an occasion when she ate fish. One can usually cure it quickly by an aperient tonic mixture. In chronic cases much good is done by five grains of quinine three times a day.

7. Here is a boy with alopecia areata. The German School until recently believed the affection to be a tropho-neurosis. Most dermatologists, however, now consider that the majority of these alopecic cases are due to a definite microbe, as demonstrated by Sabouraud. It is a very short bacillus, very similar to the microbe found in acne and in cases of seborrhœic eczema. This microbe may exist on the scalp and do no harm, but if it gets into the hair follicles it produces alopecia areata. Sabouraud produced a form of alopecia in rabbits by inoculation with it. The majority of cases will give you a history of seborrhœa and scurviness of the scalp for some time before the

Digitized by Google

onset of the alopecia, and they will say that all of a sudden patches of hair have begun to fall out. Temporal baldness is generally due to seborrhœa which has been neglected. In the majority of cases it could, no doubt, be stopped by treating the seborrhœa. Some years ago Mr. Jonathan Hutchinson brought forward the theory that a good number of these cases were the sequelæ of tinea tonsurans. I believe that Mr. Hutchinson based his theory on the case of a doctor, who came to him with a patch of alopecia areata at the top of his head, and stated that when he was a boy he had had a patch of tinea tonsurans on that very spot. Other cases with a similar history that he had come across no doubt came to his mind, and he came to the conclusion that alopecia areata was a sequela of ringworm. He believed that in the cases where no such history was obtainable the people had forgotten that they had had ringworm, or would not admit that they had had it. I took the trouble to examine into the history of many hundreds of cases of alopecia which had been under my notice, both here and elsewhere, and I found more of them gave a history of previous tinea than I expected. I now believe that some of the cases may have previous ringworm as a cause. On the whole, however, I look upon alopecia areata as a condition which may be produced in several ways—by a trophoneurosis rarely, by the poison secreted by the fungus of ringworm sometimes, but most often by that formed by the microbe which M. Sabouraud has demonstrated.

I nearly always order the same ointment for alopecia areata as I do for ringworm: carbolic and salicylic acids (3j of each to the ounce) well rubbed in. In addition to that I order a stimulating lotion to be used once a day, containing three drachms of strong liquor ammonia, half an ounce of turpentine, one and a half to two ounces tincture of cantharides, in ten ounces of distilled water with eau de cologne, or something of that sort. It is at the same time a stimulating and a cleansing lotion. Sometimes I add to it a drachm of red oxide of mercury and a little glycerine. It is also useful for ordinary seborrhœa and the falling of hair which so many ladies complain of. An excellent ointment for seborrhœa is one containing fifteen grains of red oxide of mercury, the same amount of salicylic acid, and any ointment substance you like. In all cases of alopecia areata, I also rub in Burts' fluid once a fortnight.

8. The next patient I show you is a rather old woman with a severe condition of eczema, in whom the skin has been left rough and hard. She has the eczema in the usual positions on both legs and arms. In the leg there is a condition of varicose veins in addition. It is important when the leg is in this condition to keep it well bandaged. In this case I am having the skin well bathed, night and morning, with a very weak antiseptic lotion, containing creolin or liquor carbonis detergens. In the inflamed stage, such as this, the applications must be very weak indeed. If you use creolin there should not be more than five drops to ten ounces, and the addition of a very small amount of acetate of lead to the lotion is advantageous. This lotion should be dabbed on night and morning, and a protective healing ointment put on afterwards. The ointment we are using here contains twenty grains of oxide of zinc, ten grains acetate of lead, twenty grains of the ointment of nitrate of mercury, ten grains of calomel, and vaselin one ounce. A very serviceable ointment for the later stages of eczema contains half a drachm of liquor carbonis detergens, ten grains of ammoniated mercury, and one ounce of vaselin.

9. Here is a young woman with psoriasis, somewhat inflamed. It is in the positions usually mentioned in the books—particularly on the knees and elbows. At certain examinations cases of psoriasis without the scales are sometimes shown to the candidates; in this instance you see that the patches are quite smooth, and of a dark copper tint, the scales having been removed by the treatment with an ointment containing one drachm of creolin, ten grains of salicylic acid, and ten grains of ammoniated mercury to one ounce of vaseline. For rubbing in her head she is having ammoniated mercury one drachm, soft soap three drachms, vaseline four drachms. I expect that the spots on her head will be cured quicker than those on the legs. If the eruption on the legs and arms does not soon disappear, I shall apply a chrysarobin paint, containing forty or fifty grains of chrysarobin, salicylic acid fifteen grains, and liquor gutta-perchæ in chloroform ʒj. Chrysarobin in the form of an ointment is objectionable, not only on account of its staining the linen so

Digitized by Google

much, but because, if accidentally carried to the face, a severe erythema may be set up.

10. Here is a curious case of lichen planus in a youth, who is rather a young subject for it. Very common situations for the eruption are on the wrists and on the fronts of the legs, and symmetrically inside the knees; it may, however, be present anywhere. It consists essentially of small, flat, somewhat angular papules, polished, and purplish or brownish red in colour. The papules here are rather smaller than in lichen planus usually. There is nearly always extreme pruritus, but in this case the itching is less marked now as the result of treatment. He is having a creolin bath, and rubs in a creolin and mercury ointment afterwards. He is also taking arsenic. This is one of the diseases—pemphigus is another—in which, I think, arsenic may be sometimes effective. I have little or no faith in arsenic in cases of psoriasis.

11. Here is a man whom I have had brought up from the ward. He came to the hospital nine weeks ago with an extraordinary pigmented condition of his legs and body. Here is a photograph of the condition taken by Mr. Bassano (Fig. 1). The patient had some difficulty in walking because of loss of power in his limbs. His hands were not affected at first. It appears he had been some three or four weeks in an infirmary. He says the skin was covered with "things like currants." On the leg may be seen a bleb, which may be taken as agreeing with that history. I considered it to be a case of pigmentation after pemphigus treated by arsenic. We have since got a history from Dr. Parsons at the infirmary, confirming that opinion.

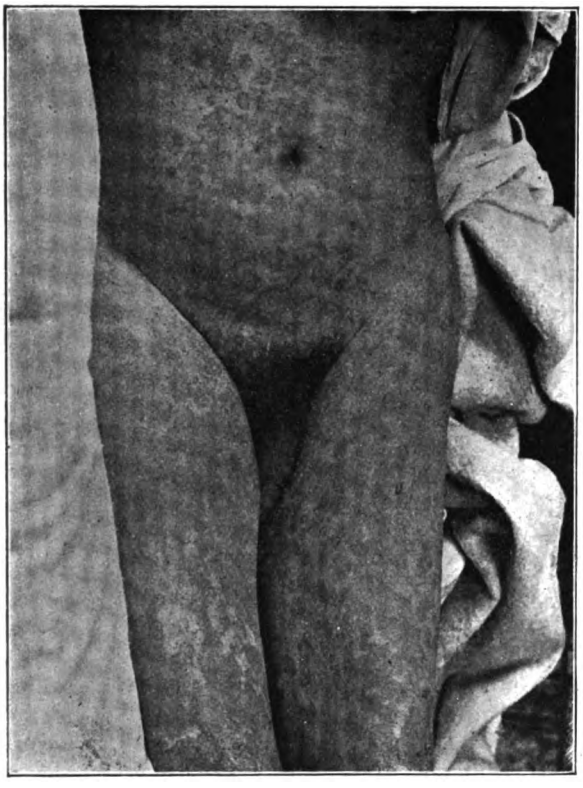


Fig. 1.—Arsenical pigmentation after pemphigus.

Fig. 2.—Lichen scrofulosorum.

It appears that in July he was admitted to Fulham Infirmary with extensive pemphigus, and was at once put on arsenic in increasing doses. The pemphigus disappeared, but the pigmentation was left. Since he has been here we have diminished his arsenic, and when we did that the blebs reappeared. On renewing the arsenic the pigmentation has reappeared, but the blebs have nearly all gone. The difficulty in walking was no doubt due

Digitized by Google

to a peripheral neuritis caused by the arsenic. We are now giving him cacodylate of soda, which is an arsenical preparation, that is supposed not to have the effect of producing such symptoms.

12. This young girl came to the hospital in June, when she had a very curious lichenous eruption on the back and chest, with small hard plugs in the follicles, and also these very ugly ulcers under the arm (Figs. 2 and 3). The history was that she had had an enlarged gland under the neck, and that a sore formed there, the scar of

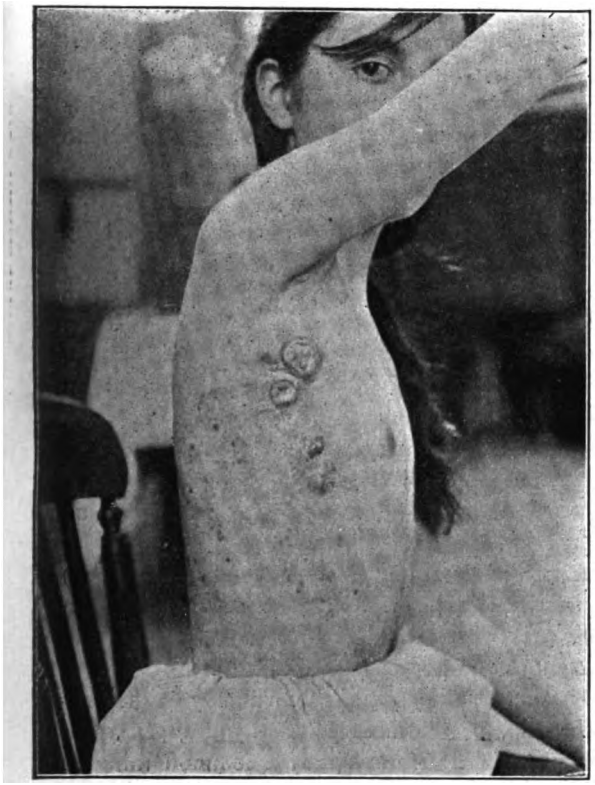


FIG. 3.—Lichen scrofulosorum.

which you see. Subsequently other tuberculous ulcers formed, and then this eruption on the back and chest came out. It is a well-marked case of lichen scrofulosorum. We soon cleared off the eruption by ointments, and now she has only slight pigmentation of the skin remaining. But the tuberculous ulcers would not heal, and accordingly Mr. Bidwell has recently scraped them out. He found very large glands in the axilla infected, and removed them. There is some little suppuration taking place, but she has no high temperature. Lichen scrofulosorum, especially in such a marked degree, is most rare; indeed, this is almost a unique case. One can very easily remove the eruption by simple tar or other ointments. In this case she had an ointment containing liquor carbonis detergens and a little cod liver oil rubbed in. I have, of course, put her on Parrish's food, with nutritious diet, etc.

W. F. CHENEY discusses the manifestations of rheumatism in children, with notes of a number of cases. As to the manifestation in the joints, they are described as less severe than those which occur in adults. The swelling is less, and the redness is often missing entirely. There is a stiffness in the joints and resistance to motion, pain when used, and disinclination to exercise. The symptoms are more apt to be limited to one joint, and the constitutional disturbances are very slight. Temperature rarely rises to 102°. The sweats so common in adults are not seen in children. Care must be taken to distinguish the pains from the so-called "growing pains." It is especially to be noted in connection with rheumatism in children that the endocardium is so frequently affected. Much has been said of the relation between rheumatism and chorea. In twenty-nine cases of the author's series, twenty-seven showed no history of rheumatism. He believes that this may be assuming that some joint manifestations really occurred previous to the chorea, or that the chorea was the first manifestation of a rheumatic series. He records two cases showing the relation between one form of purpura and rheumatic manifestations in the joints. He mentions the subcutaneous nodules at times found in the fibrous tissue as an occasional manifestation of rheumatism in children, and speaks of the grave significance of this. He mentions the relation of tonsillitis and rheumatism as remote, and he does not believe that it should be spoken of as a rheumatic manifestation at all.—*Philadel. Med. Journ.*, September 14th.

Digitized by Google

A CLINICAL LECTURE
ON
CARDIAC PAIN AND ITS TREATMENT.

Delivered at the Brompton Hospital for Consumption and Diseases of the Chest, July 18th, 1901.

By ARTHUR LATHAM, M.A., M.B.Oxon., M.R.C.P.Lond.,
Assistant Physician to the Hospital and to St. George's Hospital.

GENTLEMEN,—I do not want this afternoon to discuss the paroxysmal pain of angina pectoris, whether true or false, although I must, to some extent, touch upon it, nor the pain sometimes associated with acute disease, such as acute aortitis or pericarditis, so much as I want to consider the more or less chronic conditions which may be associated with pain in the region of the heart.

This pain has various characteristics; it may be chronic or acute, localised or diffuse; it is sometimes limited to a feeling of oppression over the region of the heart; on the other hand, it may be a definite pain which spreads to the back or down one or other arm. It may be due to many causes, some of which I propose to bring under your notice this afternoon. Thus it may arise from (1) purely local conditions, such as intercostal rheumatism, intercostal neuralgia, or intercostal periosteitis. The diagnosis of these local conditions is usually a simple matter, and rests largely on the exclusion of certain symptoms, such as dyspepsia, and on the absence of any fever or physical signs, such as a cardiac murmur. Intercostal rheumatism is associated, as you know, with tenderness in the intercostal muscles, intercostal neuralgia with points of tenderness along the intercostal nerves. Intercostal periosteitis, on the other hand, is usually associated with a history of syphilis, and often with some definite thickening of the periosteum.

Again, pain referred by the patient to the cardiac region may be due to (2) acute or chronic conditions of the lung. In advanced pulmonary tuberculosis patients often complain of pain in the region of the heart. This may be due to contractions of the lung, and consequent displacement of the heart, or it may be caused by definite pleurisy; in both conditions this symptom may be, to a large extent, relieved by suitable remedies, more especially by strapping or by plaster applied in strips.

The commonest cause of pain in the region of the heart is some disorder of the stomach or intestines—what is usually called (3) heartburn or cardialgia. This heart-burn is so commonly associated with gastric disturbance, that many patients who are the victims of a disordered digestion are convinced that they are suffering from disease of the heart, and I should be afraid to say what proportion of people come to this hospital—as you know a hospital for diseases of the chest—complaining of an uncomfortable sensation in the region of the heart, which is, in reality, due to dyspepsia. The cause of this pain is readily diagnosed, because it has a definite relation to food, and is usually associated with other dyspeptic phenomena. Moreover, when the digestion is at fault, this symptom is readily relieved by suitable diet and proper remedies. It is, however, important to remember that cardialgia comes on not infrequently in the course of heart disease, although it in itself has no relation to the heart. A man with cardiac disease is more liable than a healthy man to suffer from pain which is dependent upon some dietetic error, so that we must not forget that this symptom may be due to disorders of digestion even in patients suffering from heart disease.

Cardiac pain is frequently complained of in (4) neurasthenic and hysterical conditions, and in such cases yields to general treatment.

Another group of cases I want to call your attention to is (5) the submammary pain complained of, for the most part, by young girls. In anæmia and in chlorosis it is a common thing to find a pain in the region of the heart below the left breast, which is associated with definite tenderness over the cardiac apex. If you press on the apex of the heart, you elicit a considerable amount of pain. This symptom is found in anæmic girls, and in girls who are suffering from some genital disturbance, such as irregular menstruation, and in this latter class there is in addition definite tenderness in the region of the ovaries, more especially the left ovary. In a case of pure anæmia, rest, fresh air, good tood, and some form of iron will, in the vast majority of cases, rapidly cause all symptoms and distress to disappear. In cases where anæmia is not marked, and where the

Digitized by Google

generative organs are at fault, the pain is not relieved so readily, and in these cases I find the best treatment is counter-irritation—that is to say, repeated flying blisters in the submammary region, and in the region of the left ovary. This measure, combined with a certain amount of moral persuasion, will eventually, though not so rapidly as in the anæmic class, get rid of the pain.

Another group of cases in which this cardiac pain is complained of is associated with (6) cardio-vascular conditions—that is to say, cases of aneurysm or atheroma of the aorta, cases of definite heart disease, such as disease of the aortic valves, and less commonly disease of the mitral valves. Again, this symptom is often present in the cardiac failure which may arise in the course of heart disease or from over-exertion; it occurs, for instance, in the rapid dilatation of the heart brought on by some exhausting exercise, such as climbing the Alps in an untrained condition; similarly it is found in the case of a man, say with mitral regurgitation, who attempts to do too much, and who, in consequence, gets rapid dilatation of the heart. It is also associated with an adherent pericardium, and here must be largely due to the adhesions of the pericardium interfering with the working of the heart.

In the majority of these cardio-vascular cases the pain which is referred to the heart may be due to two causes, acting together, or independently of each other. It may on the one hand be due to an affection of the nerves, either functional or organic, such as the involvement of the nerves in some inflammatory trouble. In atheroma, for example, the aorta is in a condition of chronic inflammation, which may spread to the cardiac plexus and so involve the nerves. The second cause is dilatation of the heart. If the heart is put to too severe a strain, it is working against an increase of pressure; this increased pressure acts chiefly on the left ventricle and causes pain, especially when the cardiac muscle is the seat of myocarditis or fibroid degeneration, either by setting up a condition akin to spasm, or from mere stretching of the muscle and nerves.

I take it that pain from dilatation is chiefly due to increased pressure; a view which is supported by the fact that in mitral regurgitation this symptom is not so commonly met with as in aortic disease, probably because the mitral valve is patent, and the increased pressure in the left ventricle is in this way relieved to a certain extent. Again, in true angina pectoris associated with aortic disease the paroxysms become less marked if the patient happens to develop a mitral lesion; the paroxysms in such cases may be severe and frequent, but if the patient develops mitral regurgitation not only does the pain become, as a general rule, less severe, but the attacks are not so frequent.

There is another cause of this cardiac pain, which is really due to the cardio-vascular system. I refer to what we call the toxic group. Substances such as tobacco, tea, or coffee may act in this way; they may produce irritation with or without definite pain in the region of the heart, or they may cause attacks of pain which closely simulate those of true angina. The toxins of the specific fevers, more especially influenza, are also causes of this cardiac pain. It is not an uncommon thing for a patient to develop, some time after influenza, attacks of pain in the region of the heart which may become more or less chronic. The toxins probably act upon the nerves of the heart and in that way cause neuralgia.

A good deal has been written on the presence or absence of "pressure spots" in connection with cardio-vascular disturbances. It is possible in many of these cases to elicit definite tenderness in certain spots, especially in the second, third, and fourth intercostal spaces. These spots of tenderness vary much in their distribution, and in many cases are entirely absent. Personally I have no faith in them either for diagnostic or for prognostic purposes. Excellent work has been done with regard to the areas of distribution of pain in these conditions. Head and Mackenzie have come to the conclusion that in heart disease, especially of the aortic type, the pain is along the second, third, and fourth dorsal areas, whilst in true angina it is distributed over the fifth, sixth, seventh (and sometimes the eighth and ninth) dorsal areas, and is always present in certain of the cervical areas.

I have here some ten or a dozen cases, which I hope to demonstrate at the conclusion of this lecture. These have been carefully selected with the view of demonstrating the various causes of the symptom we are discussing. As our time is limited, however, I must for the present confine my remarks to those of them which come under the cardio-vascular group.

The first case I shall show you to-day is that of

Digitized by Google

a man who was a soldier in India for a considerable number of years; he is forty years of age and is in a very anæmic condition; his present occupation is a light one, that of night watchman. He came to me some time back complaining of pain in the region of the heart, and on examination of the heart I found that he had a double aortic murmur. There is evidence of atheroma, due no doubt to his previous occupation, and with that atheroma there is probably some implication of the cardiac plexus. The pain is behind the left side of the sternum, and radiates towards the back. I took him in at St. George's Hospital and gave him absolute rest in bed for a week, and small doses of arsenic, with the result that he has lost his pain.

The second case is that of a man, J. H—, who has complained of palpitation and pain in the chest for four years. The latter is chiefly in the region of the apex and behind the sternum. You will find he has aortic regurgitation. His case is similar to that of the other man, but is complicated by a disordered digestion, and I believe his pain to be largely dependent upon the condition of his stomach; in any case, it is becoming less under a course of treatment with stomachic remedies, combined with attention to his diet.

The next case is that of a young girl from Plaistow, æt. 17, who has had heart disease for two years. She has complained for the last few months of pain in the left side, which radiates to the back. Her heart is very much enlarged; the apex-beat is considerably outside the nipple line, and on auscultation you will find that the first sound at the apex is loud and booming, and accompanied by a systolic murmur. This is a case of mitral disease with an irregular action of the heart; the pain is largely due to the tumultuous action of the heart, but is yielding to rest and cardiac tonics.

The fourth case is that of a man æt. 18. He is a gardener from Cheshire, who came up some time ago suffering from mitral stenosis. He first complained of pain two years ago. I saw him lately, when he stated that the pain in the chest had been more severe during the previous week or two, especially in the region of the heart and left axilla, and that it had been brought on by excessive exertion. He has the ordinary physical signs of mitral stenosis and mitral regurgitation, and I take it his pain is due partly to the dilatation which has followed, and partly to the irregular action of the heart.

The next case is that of a boy, A. T—, who is excessively anæmic. He came originally to me some years back, at the Victoria Hospital for Children, after the third or fourth attack of chorea. At that time he had developed symptoms of cardiac trouble, and I warned his mother that he must not be allowed to do as much in the way of sudden exertion and running about as other children, and she has to a certain extent looked after him. He has been brought to me again suffering from mitral regurgitation and stenosis, and complaining of pain over the region of the heart, which is probably caused in the same way as in the last case.

I now come to an interesting group of cases, of which I have had four in the last two months, three cases in men and one in a woman. All of them came complaining of feeling unwell, of being unable to do their work, and of constant pain behind the sternum, which radiated in three of the cases to the back. In only one of the cases have I been able to find, after a very careful examination, any physical signs pointing to any particular derangement of the heart or vessels. In one I found a small patch of dulness, somewhat larger than a square inch, on the left side of the dorsal vertebræ, associated with an accentuated aortic sound in front. I was convinced that these patients had definite suffering, and as I could not discover any adequate cause, I sent them on to the X-ray department. They have all been returned as having a slight dilatation of the descending arch of the aorta. With regard to the patient who had an accentuated second sound over the aortic area, Dr. Blacker, who kindly examined the cases for me, wrote that he finds some dilatation of the descending arch, not sufficiently pronounced, however, to be described as an aneurysm. A week later Dr. Blacker reported that the dilatation was much more marked on the left side of the arch, and that, on examination with the screen, pulsation was visible.

The next case is that of a man æt. 47, with no history of syphilis and no physical signs. Dr. Blacker wrote that there is a small aneurysm of the descending aorta, and that pulsation is visible when the screen is used. This condition was confirmed a fortnight later.

In the case of the woman there is the diagnosis

Digitized by Google

by the X rays of a dilation the size of a five-shilling piece in the course of the descending aorta.

In none of these cases was I able to make a satisfactory diagnosis until Dr. Blacker came to my aid with the X rays. It would, I think, be unwise at the present moment to lay too much stress on the X rays in the matter of diagnosis of chest diseases, but there can be no doubt that they are of great value to us when checked by the more ordinary methods of investigation, and that in some cases where the ordinary methods have failed, the X rays are capable of giving us much important information. In my series of cases it is probable that we are dealing with an atheromatous condition of the aortic arch, and that there is a little bulging at one place more than elsewhere. The atheroma in itself would be sufficient to account for the pain which incapacitates these individuals if the inflammation of the aorta implicated the cardiac plexus.

As I have said, these various forms of referred cardiac pain may consist either of (1) a sense of unpleasantness, or (2) of definite pain, and the cause of this pain may usually be arrived at by a careful consideration of the facts of each case, and by a careful physical examination, combined with the exclusion of such causes as dyspeptic troubles and the like. Further, the great majority of cases yield to an appropriate course of treatment. A certain number, however, develop attacks of paroxysmal pain closely allied to the paroxysmal pain of true angina, that is, develop the paroxysmal attacks of false angina; whilst some, especially the aortic regurgitant group, may even merge into true angina itself. We therefore have to face the question, How can we diagnose such attacks of pain from those of true angina? Until there are attacks of paroxysmal pain combined with the fear of death, the question of true angina does not, as a rule, arise. When the attacks are paroxysmal and associated with fear of death, then we have to consider whether the patient has true angina or not.

What are the distinguishing symptoms of true angina? In the first place true angina is an excessively rare disease. In the second place it hardly ever occurs in women. It is almost invariably found over the age of forty-five. The attacks of pain are always paroxysmal, and are almost invariably associated with an overwhelming sense of impending dissolution; the pain is not so commonly associated with vaso-motor phenomena as it is in cases of false angina, nor is it associated with hysterical symptoms. Thus, a man with a history of hysterical symptoms, such as spasm of his glottis some years previously, is probably not suffering from true angina pectoris. In cases of true angina the pain is not associated with much movement on the part of the sufferer. The patient with true angina will lie quite still, huddled up in bed, and nothing will persuade him to move. The victims of false angina, especially when marked hysterical symptoms are present, perambulate the room crying out loudly for relief. In true angina the attack is usually brought on by some definite indiscretion, either by too much exercise, —that is to say, a man does too much, for instance, jumps a gate or exerts himself too much in some other way—or by some dietetic error which gives rise to an attack of flatulence.

The above is what Osler, who has written an excellent work on angina pectoris, would call a "glib list of symptoms." It is, however, an important list, and often affords us much assistance. We must, however, remember that in diagnosing true from false angina we are dealing with a most difficult subject, and can only hope to arrive at a correct conclusion by the most careful consideration of all the aspects of each individual case.

I have already spoken with regard to the treatment, in certain instances, of the non-paroxysmal pain referred to the region of the heart, but in all cases the essential thing is that the treatment must be dependent on the cause. If the pain is due to the heart-burn following dyspeptic disturbance we must attack the digestive organs. The pain dependent on dyspepsia may be relieved by suitable means; for example, by a careful dietary, by bismuth and hydrocyanic acid and other stomachic sedatives, together with an occasional mercurial purge. A definite attack of pain may be relieved by a mixture, such as Sp. Æther. Co. and Sp. Camph. Co. āā ʒj in Aq. Menth. Pip. ʒj. If this is not successful we must have recourse to small doses of morphia.

Local conditions, periosteitis, intercostal neuralgia, and rheumatism are often relieved if we support the parts by strapping, or by plasters of belladonna, and the like. I would specially mention that if the plaster is applied in strips it is more efficacious than a piece of plaster four by five

Digitized by Google

inches. Where the cardio-vascular system is affected I believe the pain is often due to dilatation of the ventricle, or to strain following on increased work, and that gives us a clear idea of what we should do for this class of patient, viz. that we should in the first instance give them rest. Careful instructions must be given as to the general health. If these, together with rest, do not relieve the pain, we may apply belladonna plaster, or better still flying blisters, constantly repeated, to the position of the pain. As far as internal remedies are concerned, there are only two drugs in my experience that have any effect at all upon the pain found in cardio-vascular conditions other than dilatation, and those are potassium iodide and arsenic. In aortic regurgitation or atheroma there is no drug which has anything like the power of potassium iodide in from ten to fifteen-grain doses. If after a fair trial, extending, if necessary, over some months, you find the pain is not becoming less, then try arsenic. My plan is to give the majority of these cases potassium iodide in the first instance. If this drug combined with rest does not relieve the pain, then I give arsenic, and alternate it with potassium iodide. In dilatation we must have recourse to digitalis and other cardiac tonics. During an attack of definite pain in the cardio-vascular cases rapid stimulants and carminatives are called for, such as ether, ammonia, and alcohol. If these do not relieve the patient, you will find that leeches, which act so extremely well in the pain of pericarditis, are probably our best remedy, especially when combined with a large dose of calomel. If leeches do not bring relief, then we must have recourse to morphia.

Insanity in Women associated with Pelvic Disease (*Ann. of Gyn. & Ped.*, February, 1901). —Dr. W. O. Henry says, "A large majority of all insane women have some pelvic disturbance as an important, if not a chief, causative factor," and early and radical treatment of those so affected is highly important.

Morel states that the brain is the seat but not always the cause of insanity, and Kohlberger, More Madden, Kræmer, Skene, Rohé and others hold the same views. The latter considers that puerperal insanity, melancholia, and simple mania are the forms of insanity which are chiefly amenable to treatment of diseased pelvic organs.

Many gynæcologists will not operate upon any patient who shows any taint of insanity, as they fear to precipitate an attack of mania, but this is a great error on their part, as the operation is far more likely to cure than aggravate the condition.

Dr. Henry appends a list of some sixteen cases in which he operated with excellent results. He considers that gynæcologists who operate upon the insane would get better results if they were not so conservative.—*Montreal Medical Journal*, August, 1901.

The Therapeutics of Whooping-cough.—T. J. Mays advocates the application of counter irritants over the region of the pneumogastric nerves in the neck, and states that, in his experience, this method is the only one which leads to any notable amelioration of symptoms. His method is thus described: Trace the pulsating carotid artery from behind the angle of the lower jaw to the clavicle on both sides of the neck. This will be a landmark for finding the pneumogastric nerves, which lie in close proximity and slightly behind the carotids. Gentle massage and kneading of this region of the neck every hour or two yield beneficial effects in many cases of this disease. The application of a strip of mustard-plaster, about two inches wide, from the angle of the lower jaw to the clavicles on each side of the neck two or three times a day, until the effects of the mustard are evident, is almost sure to cause amelioration of the spasmodic cough. Equal parts of gum camphor, chloral hydrate, and menthol, applied over this region, are also very useful. Painting the same area with tincture of iodine twice a day, until irritation of the skin is produced, is a beneficial procedure. Finally, in very stubborn cases the hypodermic injection of silver nitrate over the vagi must be resorted to in accordance with the following plan: Lift the skin over the vagus between the thumb and the forefinger of the left hand, introduce the hypodermic needle just under the elevated skin, and inject five minims of a two and a half per cent. solution of cocaine hydrochloride. Detach the syringe from the needle and allow the latter to remain in the puncture. Wash out the syringe with water, draw a two and a half per cent. solution of silver nitrate into the syringe, attach the latter to the needle, and throw in from three to six minims of the silver solution.—*Medical Record*, September 14th, 1901.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 468. **WEDNESDAY, OCTOBER 16, 1901.** **Vol. XVIII. No. 26.**

CONTENTS.

PAGE

***A Clinical Lecture on some Points connected with Thrombosis.** By Sir WILLIAM BENNETT, K.C.V.O., F.R.C.S. 401

***A Demonstration of Surgical Cases at the Hospital for Sick Children.** By BERNARD PITTS, M.A., M.C., F.R.C.S. 405

***With Dr. Galloway in the Skin Department of Charing Cross Hospital.** 410

** Specially reported for The Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers,* 22½, *Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Cheques and Post Office Orders should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C."*

Terms of Subscription, including postage, payable in advance: for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

The Index and Title-page for Vol. XVIII will be published with the next issue of THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

SOME POINTS CONNECTED WITH THROMBOSIS.

Delivered at St. George's Hospital

By Sir WILLIAM BENNETT, K.C.V.O., F.R.C.S.

GENTLEMEN,—There are few things more instructive for clinical purposes than the comparison of two or more cases illustrating different phases of the same disease, and there are few diseases, I suppose, which have more variety in phase than venous thrombosis, by which, of course, I mean the blocking of a vein by clot, and so far as we are concerned to-day the blocking of a normal vein, and not a vein which is abnormal in consequence of varicosity. There have been lately two cases in the hospital which are admirable examples of different phases or types of this condition, which we call thrombosis. Both were of their kind very exaggerated instances. Both were serious cases; one indeed extremely so. One of the patients is still in the Wellington Ward and the other was in the Harris Ward. The former is a woman, forty-nine years of age, and married. She came to the hospital on account of a femoral hernia which had given her some trouble, and she was very anxious to have it dealt with by the radical operation. She was operated upon in my absence, but I am assured that there was nothing peculiar about the case; the sac contained no blood, as is very often the case in femoral hernia, nor was there any condition whatever suggestive of coming trouble. The operation was performed in the usual way with the ordinary precautions, the patient being in excellent condition upon her return to the ward. There was a little rise of temperature after the operation, as so often happens in these cases, but the rise of temperature as a rule subsides in the course of the succeeding twenty-four hours, a fact which should be common

Digitized by Google

knowledge to all of us. In this patient the fever did not subside but remained high, and a few days later the temperature began to rise rather ominously; the patient was indeed never quite comfortable generally after the operation, although the local conditions were perfect. On December 2nd (the operation having been on November 21st) a very rapid rise in the temperature took place, which reached a point between 103°F. and 104°F.; there were no rigors. As she complained of some pain in the left leg, it was found that the left lower extremity was swelling, and examination showed pretty clearly that she was suffering from complete thrombosis of the saphena vein, as well as from commencing thrombosis in the femoral vein. She now became very seriously ill; the thrombosis extended rapidly upwards, and as it extended into the belly the opposite limb became similarly involved. The temperature still continued very high indeed, and she began to be troubled with one of the most distressing symptoms in these cases of thrombosis, that is to say abdominal distension. This was by degrees overcome, but still she remained very ill and a high temperature persisted; she was obviously in an extremely septic condition. Her face had the aspect of a person suffering from septicæmia. She had one or two rigors, which were not very pronounced, but sufficiently so to be termed rigors. On December 27th (nearly four weeks after the onset of the thrombosis in the lower limb) she had a sudden attack of difficulty of breathing of which she almost died, the result of pulmonary embolism. Later on patches of consolidation showed themselves on the right lung, proving pretty conclusively that the course of events which I have mentioned had taken place. For a long time she looked very ill, and she is still very ill. A large sloughing mass has come away over the sacrum, exposing the bone and leaving a great cavity. Her temperature has now (March 12th) sunk lower than it has been throughout the illness, and she looks as if she might possibly recover. Such is a brief description of her case; time does not admit of going into details more minutely. Here, then, is a typical case of septic thrombosis followed by embolism occurring in a patient after operation. How much the operation had to do with the occurrence of thrombosis, and how much it had not, I am not in a position to say; sepsis arose from some cause or other which is not apparent. I want you to particularly note that the embolic attack which occurred in this patient's case, did not happen until three weeks after the formation of the thrombus, and not until the progress of the thrombus had stopped. In point of fact, at the time of the occurrence of this embolism the thrombus was commencing to diminish and dissolve away. That is the point I wish you to particularly keep in mind for the present.

In the other case to which I have referred the patient was also a woman; she was twenty-seven years of age. She was admitted on January 16th into the Harris Ward, on account of severe appendicitis with a high temperature. We operated at once, opening a small abcess in connection with the appendix; as far as this was concerned all went well. The patient was intensely anæmic, and those of you who will remember to have seen her, will recollect the extraordinary translucency of her complexion, and its delicate waxy colour, which is peculiar to a certain class of anæmic people. Some of you may also remember that she had quite a high colour in each cheek. It is well to bear in mind that anæmia is not always associated with absolute want of colour in the cheeks, or want of colour in the skin generally. A patient may be extremely anæmic and yet have a high colour over each cheek. That was the type of this woman's anæmia. Her temperature, which was high at the time of operation, came down in the ordinary way, and though the case was a very severe one, she did very well. She had a little abdominal distension immediately after the operation, which of course she would be likely to have under the circumstances. A week later she was going on perfectly well and feeling quite comfortable, although a little annoyed at being kept so closely to her bed. At the end of this time it was found, quite accidentally, that one of her lower limbs was swollen; at the time of the discovery of the swelling she had not complained of pain, nor was she conscious of anything being wrong. You will remember in the other case that the symptom which called attention to the swelling was the feeling of acute pain by the patient. On examination of the swollen limb it was soon ascertained that there was extensive thrombosis in the left lower extremity, which finally involved the femoral and iliac veins. The thrombus was, as I have already said, discovered almost

Digitized by Google

accidentally on the seventh day after the operation. On the day following the discovery of the thrombus, and whilst the clot was rapidly extending up the thigh towards the groin, a sudden attack of cardiac pain with intense difficulty of breathing occurred, from which she very nearly died. A clot had passed through the heart and lodged in the lung, giving rise to the usual symptoms following such a circumstance. Here, then, was a case of thrombosis occurring after an operation in which the patient was doing well, and free from any suspicion of sepsis, followed almost immediately by embolism. In the other case the embolism did not occur till very much later, until, in point of fact, the thrombus had commenced to disappear. In the second case the embolism occurred during the process of the growth of the thrombus. These are two very different conditions, and their importance is great. The thrombus in the first case was the result of septic conditions; in the second the clotting was purely passive. The second patient's blood clotted in her veins because she, being week and intensely anæmic, had been called upon to bear the shock, of what was to her a serious operation, which lowered her vitality considerably. It is well to bear in mind that all patients of the anæmic type are very prone to blood-clotting, and that a certain percentage of them, if placed in bed and kept absolutely quiet after an operation or accident, by which their already feeble physique is still further reduced, are very liable to get thrombosis, more especially in the lower extremities, although the clotting may occur in other parts. When passive clotting like this occurs, it shows that the blood is in a condition in which it clots easily, and unless the circulation of the venous blood is helped by muscular contraction in patients of this sort, there is a great tendency, under certain conditions, to get thrombosis in the veins. In cases of the anæmic type with passive thrombus, the clotting may involve more than one side of the body, not necessarily at the same time, but in succession. For example, in this woman, as soon as the lower limbs began to get a little better she had thrombosis of the left subclavian vein, with the resulting swelling of the upper extremity. This gave rise to very little trouble, and caused very little pain, which is singularly absent in many of these passive cases. The extent of this girl's thrombosis was far greater in the aggregate than that of the woman in the Wellington Ward; yet the latter has been ill ever since, and has in fact nearly died, but the girl, directly the clotting ceased to extend, progressed comparatively rapidly, and she has now left the hospital. The other patient is still in the hospital suffering from various septic complications, and the issue of her case is still in doubt.*

We here have two very excellent cases showing different types of venous thrombosis. There are two other varieties of passive thombosis to which it may be well to direct your attention. The first is that which is prone to follow any exhausting disease, such, for example, as enteric fever, the thrombosis following upon which is frequently passive, not septic, and the second is that which follows upon great loss of blood—an admirable example of the latter variety was afforded by a patient who was brought into the Princess Ward in the middle of the night, about a year ago. She was desperately ill, and appeared to be bleeding to death from some intra-abdominal condition, the nature of which was not clear.

I operated immediately on arriving at the hospital, and found the condition to be ectopic gestation. The local condition was removed in the ordinary way, and the belly thoroughly cleared of clot; she left the operating theatre almost moribund, but with the aid of transfusion of saline solution and copious enemata of the same fluid she rallied, and made rapid progress—no rise of temperature followed, nor did any pain occur. On the seventh day after the operation she complained of swelling of the right arm, which rapidly increased, and was followed by swelling of the right side of the neck, face, head, and upper part of the right side of the trunk—exaggerated œdematous swelling, which was quite a remarkable thing to see. She had, in fact, complete thrombosis of the right innominate vein, a very grave condition under ordinary circumstances. Recovery followed slowly but completely, and no trace of defective health was finally left. If this had been a septic case with thrombosis to that extent, she would have certainly died. A point of some practical importance arises in relation to these cases of passive thrombosis which cannot be too widely known. There is a tendency in many subjects—notably those of the anæmic kind on

* She has since recovered perfectly.

Digitized by Google

the one hand, and those of the robust "gouty" type on the other—to the occurrence of thrombosis in the lower limbs if such subjects be placed in bed, when in a condition of apparent good health, and kept absolutely quiet for a considerable period—such quiet as, for example, may be necessitated by an operation on the knee-joint, severe abdominal cases, or any other cases of severity. Thrombosis is not, for an example, very rare in certain types of patients laid up suddenly with fracture, the thrombosis occurring as often as not in a sound limb. The practical bearing of this matter is the following:—In all operations of expediency, such, for example, as the radical cure for hernia, operations upon varicose veins, etc., when performed upon patients in sound general health, who up to the time of coming under treatment have been following their ordinary callings or amusements, it is wise, and in many cases necessary, that the patients should be kept in bed for some days prior to the operation, so that the absolute rest entailed by the operation comes less abruptly upon them—a period of modified rest in the flat position intervening between the ordinary habits of life and the total rest which should follow upon the operation. I have referred to this subject from another point of view in the 'Practitioner' of August last.

I have seen many cases which illustrate the importance of this point. I have, indeed, now under my care a patient who would not submit to this period of rest before undergoing an operation for the radical cure of hernia; being a very busy man, he said he had no time for preliminary rest. He went to bed one evening, and I operated on the following morning; the operation was a severe one, necessitating absolute quiet for a fortnight. He is now suffering from what are called attacks of gout, *i. e.* he has a blocked vein first on the dorsum of one foot, and then in front of the opposite ankle; the attacks are at first quite painless, and there is no rise of temperature. These blocks are, in my opinion, not in any sense "gouty" at all, but are the result of passive thrombosis resulting from the complete rest in the flat position. He is now getting better, because he is beginning to move about a little, and the tendency to stagnation ceases. That is a practical point which is, I am sure, worth bearing in mind. It is not at all uncommon to hear of a man with a broken leg having attacks of "gout." Very often the "gout" is a purely passive blocking of a vein. Such "gouty" attacks are singularly obstinate so long as the patient is lying absolutely still, but generally rapidly disappear when the circulation is accelerated by the movements of the patient when convalescence begins.

In all cases of thrombosis, whether septic or aseptic (*i. e.* passive), no matter what the cause may be, there is a certain period in each variety at which embolism is more prone to occur than at any other time. A proper appreciation of this fact is of some importance, since the time at which embolism is prone to occur is that during which absolute rest for the patient should he most rigidly enforced.

You will remember in the first case which I have mentioned (the septic case) embolism did not occur until three or four weeks after the onset of the thrombosis, and that no embolus became detached until the thrombus had not only ceased to grow but had commenced to disappear; the embolism in fact occurred at the time of softening of the thrombus. The importance of this clinical point is as follows:—The period of danger from embolism in a *septic* case of thrombosis is at the time of softening, *i. e.* when the patient is apparently on the verge of convalescence.

On the other hand, bear in mind what happened in the second case, in which the thrombus was *aseptic*. Extensive thrombosis occurred very soon after the operation, and on the following day embolism occurred whilst the thrombus was increasing in size—a clinical sequence which illustrates admirably the fact that in aseptic thrombosis the danger of thrombosis is greatest during the formation of the thrombus, and not at the period of resolution, the exact reverse of the condition holding good in septic thrombosis. These are very practical points, which, so far as I know, you will not find emphasised in the text-books, but they are nevertheless undeniable truths unless my experience is entirely at fault. The practical bearings of these facts upon treatment are the following:—in *septic* thrombosis the greatest care should be taken to ensure absolute rest during the period of resolution; in *aseptic* cases the greatest call for rest is during the period of the formation of the thrombus. In septic cases on no account should

Digitized by Google

patients be allowed to move until the thrombus has entirely disappeared; in aseptic cases, on the other hand, there is no objection to a little movement when the thrombus has ceased to grow, and there is certainly no reason for maintaining complete rest until it has entirely disappeared, in fact in the later period some movement is beneficial.

I have recently been seeing a case which illustrated both these conditions of thrombosis. Passive thrombosis occurred in one limb, and before that was well a septic thrombosis in the other leg followed. Embolism happened in both attacks—a most remarkable thing. The patient ought to have died, but oddly enough he recovered from both attacks of embolism. The embolism in his case pursued exactly the course which I have described in these three cases to-day, the aseptic one occurred during the progress of the thrombus, the septic embolus was detached during resolution.

One word with regard to the treatment of these cases of passive thrombosis, about which there is some difference of opinion. Rest of course is absolutely necessary during the progress of the disease. Medicinally I am sure the best treatment is what is called the alkaline treatment—alkalies with excess of ammonia will do more to hasten the disappearance of these passive clots than anything else. Locally warm applications are comfortable, and perhaps to some extent promote absorption. When the veins are varicose if the application has some hardening effect upon the skin (Lot. Plumbi, for example) so much the better—when the veins themselves are normal water fomentations effect every purpose. In the late stage of aseptic thrombus massage, when used with understanding, is of great service; but by those who are without considerable experience it should not be used as in such hands it may be dangerous. The operative treatment of progressive and recurrent thrombosis is important and interesting, but time does not allow of my discussing the subject to-day—moreover I have dealt with it fully elsewhere somewhat recently.*

* 'Varix and Thrombosis.' Longmans and Co.

A DEMONSTRATION OF SURGICAL CASES AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

June 13th, 1901.

By BERNARD PITTS, M.A., M.C., F.R.C.S.

GENTLEMEN,—This boy illustrates some of the difficulties in getting rid of a tracheotomy tube. He was tracheotomised for diphtheria in the hospital two years ago, but those who have had to deal with him have never been able to get rid of the tube for any length of time, though a good many attempts have been made. Mr. Collier, who is in charge of the case, has cut down to examine the tracheotomy opening, and on the first occasion he found some granulation tissue, which he removed, and he thought that would get over the difficulty. But that did not succeed. He therefore made an incision through the thyroid so as to see whether there was any mechanical obstruction still existing. He found nothing except cicatricial tissue around the tracheotomy opening. Since then the boy has been intubated on and off several times, for a week he has had the tube in, and then it has been taken out again. Up to the present, however, the case has defeated all attempts to make the patient do without a tube. These cases are always extremely difficult; some of them I suppose are really insuperable, but I should never despair, because there is no doubt very much can be done by persistent intubation, using large-sized tubes, after carefully inspecting the part and seeing that there is no mechanical difficulty. The most ordinary cause of difficulty in leaving out a tracheotomy tube is fright on the part of the patient, and the spasm which comes on. But that can always be got over by management, and repeated attempts at leaving out the tube after gaining the confidence of the patient. But in cases where there is mechanical obstruction there is often very considerable trouble. A pressure sore sometimes comes on the front of the trachea from the riding forward of the lower end of the tube; but that has nothing to do with the subject I am referring to. The trouble arises from the pressure or the irritation of the neck of the tube, and in all the cases that I have seen the

Digitized by Google

cicatricial or mechanical obstruction has been just above the tracheotomy opening, either in the trachea or in the lower entrance of the larynx. So it is in the part above, rather than directly corresponding to any portion where the tube lies in the trachea. From the cases I have seen I should put granulations as the most frequent cause of the trouble in the early cases; but if the trouble persists, then this will probably go on to the formation of cicatricial tissue. Then you may get cicatricial tissue forming either a tubular stricture, or you may get an actual diaphragm with just a small opening in it forming at the lower entrance of the larynx. Another difficulty arises from a certain amount of kinking in the trachea, which is apt to take place opposite the tracheotomy opening when a tube has been worn a long time.

If you find that you fail in getting rid of a tracheotomy tube, and that you have eliminated all the ordinary causes of spasm, and of any granulations which are obviously flapping in the wall of the trachea; the next thing I think is to try intubation, and see if that succeeds. If it does not, then you must remove any obvious cicatricial thickening of the skin and the soft parts outside the trachea, so as to prevent that tendency for it to contract into a minute aperture directly the tube is left out, and make it a clear soft vent, and you must explore further and see whether there is any cicatricial tissue forming a diaphragm or tubular contraction, and if so it must be removed. You have to cut it away with scissors and forceps, and get it away in the most radical manner; half measures are of no avail. Having done that, you are in a position to carry out intubation. The intubation in such instances differs a little from the intubation necessary in acute cases—I mean intubation for diphtheria or for scalding. It differs in this respect, that you need to gradually work your way up to a very much larger size intubation tube than for acute cases. I should say that if you are dealing with a child of five years of age, you must gradually work up the size until you get an intubation tube that is required in the ordinary case for a child of twelve years. Until you get a tube of that size thoroughly established and worn by the child for some time, you do not advance very much in the matter. Such cases require an infinite amount of patience and time to carry out the treatment.

About the year 1890 I published particulars of a series of cases illustrating this difficulty of leaving out tracheotomy tubes, and in some of those cases the intubation tube was worn for weeks at a time; one was left in several weeks without being changed. So accustomed did the children get to the tube, that they were able to go home and come up regularly as out-patients. They ate, slept, and played perfectly happily with one of these tubes in.

When you are using one of these larger tubes you can do without a string attached, because there is then no danger of the tube slipping down. In the introduction of the tube, you must be prepared to use a certain amount of force. The very gentle manipulations which are sufficient in an ordinary acute case have to be superseded by a very gradually increased pressure until it becomes considerable, just as when you are trying to dilate a stricture of the urethra. That pressure, of course, has to be steadily maintained, and you may have to withdraw the tube a little to allow some air to go in, and then press on again until the tube is well established in its place. I will mention one case of a series which I had, because it illustrates practically most of the difficulties which one meets with, and then our resident surgeon will kindly intubate the little patient before us, so that you may see the process carried out. It is an operation which requires a good deal of practice, and one which in the ordinary routine hospital work you do not often have a chance of seeing performed.

The case I will mention first is one of a series, and it is the case I learnt most from. A child of three was admitted to Great Ormond Street in 1886, under Dr. Cheatle, with difficulty in breathing and marked stridor, and had been in the hospital for a short time earlier in the year for a similar attack, from which he recovered. Tracheotomy had to be performed on December 14th, 1886. In 1889 he was transferred to my care still wearing a tracheotomy tube, and on extending the tracheal opening upwards and partially dividing the thyroid, a pinky white band of fibrous tissue was found starting from just below the cords, and extending downwards from right to left, and forming a sort of diaphragm nearly blocking up the lower

Digitized by Google

aperture of the larynx. This was freely removed with scissors and forceps, but intubation with large tubes was necessary from time to time during the next year before a complete cure was effected. This boy went through everything in the way of treatment, including Macewen's intubation tubes, operation on the larynx, and then the O'Dywer's intubation, and then tracheotomy again, then persistent return to O'Dywer's tube, and finally he was completely cured.

There were two or three other cases which illustrated different forms of cicatricial contracture. The contracture does not always occur as a band; sometimes it is a tubular cicatricial or posterior cicatricial thickening, but always at the junction of the tracheotomy opening with the larynx opening. It is difficult to understand why in certain cases this cicatricial change should take place, but I think in some cases it was due to the tracheotomy opening being performed a little too high, involving division of the cricoid. It is important to avoid dividing the cricoid in performing tracheotomy. This division prevents the easy riding of the tube, and causes the tube to be a constant source of irritation, makes the patient restless, and the extra movement adds to the irritation.

The next point is the kind of tube. Every tracheotomy tube should have a movable collar. There is no doubt such a tube will prevent a great deal of irritation. Lastly we may consider the kind of condition for which tracheotomy is done, and particularly cases attended with scarlet fever, or scarlatinal diphtheria. Although primary tracheotomy was done on the case last narrated for some cause which was never ascertained, he had scarlet fever supervening, and I have no doubt a great deal of the difficulty was due to the prolonged attack of scarlet fever during which he had to wear the tube, and in that way a great deal of irritation was set up. In another case which I had afterwards, in which there was a very malignant form of scarlatinal diphtheria, there was a great sloughing of the tracheal wall. Several of the rings came partially away, and it was a bad wound for a long time. We had a tubular stricture resulting, which gave us a great deal of trouble, but by means of O'Dwyer's tubes, which were left in for two or three weeks at a time, he eventually got over the stricture and we were able to dispense with the tracheotomy opening. I may mention that several of these patients had tubes in for two or three weeks at a time. The operation of intubation requires plenty of practice. Extraction of the tube is sometimes difficult, especially when a long tube is used. One wants a short-necked extractor. Here I show you the O'Dwyer's tubes; and we find in practice for these cases of stenosis that it is necessary to use larger tubes than O'Dwyer recommended, and then you will avoid having them coughed out. In diphtheria I should leave the thread in position, so that the tube could be peeled out by the nurse should it become blocked by any membrane. With this boy, however, that is not necessary. The tube should be greased and warmed before insertion, and it is better to have a pillow under the neck and pass the left first finger over the back of the tongue and feel for the epiglottis, and then put your finger behind it and throw it forwards. Then the end of your finger is over the entrance into the larynx. Then you pass the tube along your finger and slip it into the larynx.

The case shown was then intubated by Mr. Fairbank, the Resident Medical Officer.

The next case is that of a little girl with a small tumour in the upper and inner angle of the orbit, which apparently has some connection with the bone. It is a dermoid cyst; and when it occurs in this position you have to be careful about your diagnosis, that is to say between dermoid cyst and meningocele. If there is transmitted pulsation in the tumour one has difficulty in making up one's mind whether it is meningocele or dermoid. But here is a case so absolutely clear that I should not have any hesitation in saying it is dermoid. We shall find the cyst has a smooth lining and contains sebaceous material, and in all probability we shall find one or two hairs.

Here is another child, who has just come in to-day with a somewhat similar condition, but in the middle line of the neck over the thyroid cartilage. This cyst is formed in connection with the thyro-glossal duct. You here get an apparently movable fluctuating swelling, which feels different to any ordinary abscess. We have a long history with it. It is almost in the middle line, and we shall find it is overlaid by muscle. No doubt we shall dissect it out without difficulty. In removing these dermoids

Digitized by Google

it is very important to take care that you get the whole of the lining wall away. If you leave a part it will give rise to trouble, and there will be a re-formation of the cyst, or continued secretion, and sometimes inflammatory trouble. I had that impressed upon me once by one of the first dermoids I saw in the mouth, and projecting into the submaxillary region. These are apt to be mistaken for ranula, and that one was treated as ranula. It was incised, and the contents came away, and it was thought that would cure it. In two or three days' time the child suddenly got œdema of the glottis from inflammatory trouble supervening. Tracheotomy had to be performed in consequence, and then the radical operation, which ought to have been done in the first place, was performed, the dermoid cyst being completely dissected out. There is no difficulty with the external incision, but it looks difficult when the cyst is in the mouth more than in the neck. It is never wise to attempt extirpation of a dermoid cyst through the mouth; it should be always attacked from the exterior. These dermoids not infrequently remain quiescent for a considerable time, and then, for some reason or other, they begin to increase in size, and occasionally the increase is due to inflammatory change, but very often it is due to simple increase in secretion from the lining membrane.

Here is a little boy who presents a certain advanced stage of cure of a large hairy mole. This mole was absolute in two thirds of the face, as shown by this photograph, and was of a very pronounced type. Of all the disheartening cases one has to tackle I think the hairy mole is about the worst. At one time we used to treat them with electrolysis, but it required a great deal of time and patience, and progress was too slow for a mole like the present one. Of course in certain situations they can be excised; but the difficulty with hairy moles on the face is lest the result of treatment should be worse than the surgical condition, and you have to be guided very much by your experience in a particular case as to what should be done. I am bound to confess that although I was sceptical about the chances of improvement in this case, a very marked improvement in personal appearance has taken place, and I think a great deal more will eventually take place. The treatment has been by removing sections at a time, and then by cutting out pieces of skin from some other part of the body, not Thiersch's grafts, but the whole thickness of the skin, in the old-fashioned way in which surgeons used to transplant prepuce or portions of skin over raw surfaces. Already very great improvement has taken place. Treatment has been carried on for seven months now by Mr. Kellock. I have seen one or two children here who were almost absolutely covered—back, abdomen, limbs, and face. In one case I was able to remove the moles from the face because they were in patches the size of half a crown, and could be excised, and parts brought together by plastic procedures; but, of course the general hairy mole of the body could not be treated.

Here is a young child with a double hernia of considerable size, with the penis almost lost in consequence of the great size of the scrotum. I suppose in this case one will have to depart from one's usual rule, and operate upon a child of such early age. It is only nine months old. I should give it as a general rule that if hernia persists after the age of three or four years, it ought to be operated upon for radical cure. I do not believe in trying trusses for a longer period than that. But in hospital one has to take into account the unfortunate epidemics of infectious disease, which may arise immediately after you have operated. In such circumstances you cannot say here is an operation which does not expose the patient to any risk, and that you can take a hundred cases and get a hundred recoveries. Though you may do a considerable number of these operations without trouble, you may do others and find scarlet fever cropping up just after you have carried them out. I think, as a rule, measles does not affect the wound very much; but scarlet fever is another matter, and there is always a great deal of anxiety when that disease supervenes. As a rule I do not operate for the radical cure of hernia below the age of two, unless there is some special reason. Here is another case of double hernia which cannot be managed by the application of any sort of truss, because the child could not wear it without suffering from excoriation. The apertures are too big and the case is too far advanced for treatment by truss. I think it is worth while running the risk I have mentioned, and doing the operation. There are many special points in connection with the radical cure of hernia

Digitized by Google

in infants. I will mention one of double inguinal hernia complicated by general ectopia vesicæ. A radical cure was performed on one side—the testicle removed. The wound was covered with gauze and collodion, and by keeping the child on the opposite side contamination with urine was avoided. When the wound was healed the same thing was done on the other side. The whole of the skin of the scrotum was now available and was glided up, and the whole of the raw surface of the bladder was satisfactorily curved in. That was about ten years ago, and I have had the opportunity of seeing the boy from time to time since. The trouble is that under this well-protected covering he gets an irritating amount of phosphates, and it is very difficult to get the concretions away from under the covering.

Here is a boy, just admitted, the subject of one of those forms of talipes in which various tenotomies have been done. He has worn splints, but a satisfactory foot has not been arrived at. It is a case in which the old operation of systematised tenotomy has been performed. Of course if soft structures only are involved, and you get the case early—speaking of those cases which require operation and cannot be treated by massage,—what you have to do is to divide all resisting structures, and it does not matter particularly what is divided so long as it is a resistant structure—it may be tendon, or it may be ligament. Whatever it is, it has to be freely divided subcutaneously until you can easily replace the foot into a correct position. In a certain number of cases, however, you cannot do that on account of the changes in the shape and position of the bones. I take it that this is one, and it is not possible to put the foot into good position. There is a good deal of riding outwards of the astragalus, and alterations about the bony connections. So I think the best result we can get is by a modified operation upon the bone. One does not care to do that in very young children, unless there is some special reason for it; but I think there is sufficient reason here to remove a portion of astragalus, or, perhaps, take a wedge out. I should make an incision and expose the bones, and then determine what is best to be done to make certain that the foot will come easily into good position, and remain in position without any forcible holding. Until you get that you will not have a satisfactory result.

Phelp's or the open operation for talipes is frequently performed at this hospital, namely, making an incision directly through all structures down to and through the ligaments, so that you wrench open the joints until you get the foot straight. You can fill in the gap by implanting redundant skin, which you get from the outer side of the foot, where a considerable fold is at your service when you get the foot into right position. You can turn that flap over and tuck it into the gap which is made. The results of the open operation are satisfactory in many bad cases, but not if there is real bony deformity. They are suitable for bad general contracture of all soft parts. I am very averse to any open operation, whether by section or by operations on the bone, in very young babies. I should prefer to wait for the open section until three or four years of age, and for operation on the bone till later than that. I think all the younger cases are best treated by persistent massage, assisted by apparatus and such subcutaneous incisions and constant stretching as may be indicated, reserving the more radical procedure of open section or bone resection for the later stage, when the foot is more fully formed.

Here is a young child which presents a nævus on the skull. It is the usual form, and the question is what should be done for it. It has also one on the arm. If the nævus on the skull were on any other part of the body one would excise it without hesitation; I think we shall get a better result, and with less after-scarring, by electrolysis, but of course it will take time. Electrolysis has to be patiently persevered in, and will probably require two or three applications of the needles. I think in this case electrolysis will cause the nævus to shrink down and will leave less disfigurement and will give a better chance for the growth of hair than if the nævus was excised. The nævus on the arm is more suitable for treatment by means of Paquelin's cautery; I should not waste time by electrolysing here; though one would try that if the patient were in that class of life in which one would require to consider the cosmetic effect. Not infrequently when electrolysis was very largely employed one used to find that some of these nævi on the face resisted treatment, and there was a type of case, especially in the cheek, which did not tend to consolidate. If you are going to get a good result from electro-

Digitized by Google

lysis in a particular case it will feel harder after the first use of the needles. In some cases you will make bubbles; but you do not get hardening, and you will always find there is a lot of mixed tissue in such a case, and that it contains connective tissue and fat; indeed they somewhat simulate hygromas. Those are the cases which are only treated satisfactorily by excision. In such cases you should lift up the flap and dissect them out. I think the cheek is one of the most common places for those which are not acted upon by electrolysis. In the cheek one has to be exceedingly careful how removal is carried out, because of the liability to damage the facial nerve. To remove the nævus and damage the facial nerve in the attempt, is a condition of affairs which is very unfortunate.

That is the best series of cases I can show you to-day, as the ward has just been reopened after one of the epidemics I have been speaking about.

PHENALGIN, ANTIPYRETIC AND ANALGESIC.—Mr. E. J. Reid, of 11, Dunedin House, London, has sent for our consideration a sample bottle of "*Phenalgin in Capsulæ Rubræ et Albæ.*" Each capsule contains five grains of Phenalgin, and the idea of putting this valuable drug up in the peculiar style adopted is much to be recommended, because it insures certain identification. It enables the practitioner to prescribe the genuine preparation in a proper condition, and secures the necessary quality of prompt solubility. It is quite accurate to state of these Phenalgin capsules that when used for antipyretic and analgesic effect symptoms of depression are never observed. This enables the drug to be employed in chronic cases of neuralgia. Phenalgin is an ammoniated synthetic coal tar product, in which the ammonia is so combined that it admits of prompt liberation when the drug is ingested, with the result that its diffusible stimulating effect prevents any undue cardiac depression. For the treatment of dysmenorrhœa, and more particularly in the functional types of this condition, Phenalgin is most beneficial, and the readiness with which it can combine with other remedies, further enhances its utility. In the many instances where it is expedient to avoid the exhibition of opium Phenalgin undoubtedly is a most efficient and desirable substitute.

WITH DR. GALLOWAY IN THE SKIN DEPARTMENT OF CHARING CROSS HOSPITAL.

LADIES AND GENTLEMEN,—I have in the room to-day for your inspection cases selected from my out-patients during the last two or three days. Some of them are of special interest, and others are perhaps of interest more from the point of view of treatment, which after all is the most important matter.

The first is this young woman, who has suffered from *lupus vulgaris*, and I want to draw your attention to her specially for this reason—that she was under medical observation for something like three or four years before she came to the hospital with the patch of the disease, of which you see the mark now on her right cheek, and which had been pronounced to be sclerodermia. I suppose the reason for this diagnosis was that it was one of those very superficially infiltrating tuberculides of the skin which are sometimes a little difficult to diagnose, but which respond most admirably to treatment. When she came to the hospital we recognised that instead of simple atrophy of the skin, the patch of disease was infiltrated, that there was some new growth—that there was a cellular infiltration affecting the cutis. On looking at the various features of the case there could be no doubt that we were dealing with a case of true tuberculosis—of lupus vulgaris. As it was such a superficial case I thought the best method of treatment would be the one which is used with so much success in Paris, and which gives most excellent results, namely, the treatment by multiple scarification. This treatment consists of numerous very superficial incisions, both parallel and crosswise, of the inflamed surface, so as to break up the whole of the diseased tissue, and at the same time destroy the blood-supply, producing in that way a fine atrophy of the surface of the skin and removing as little tissue as possible. I did that, not by any of the special knives consisting of many blades together, which are exceedingly difficult to keep clean, and not at all easy to make perfectly sterile, but by means of a fine scalpel. In the first instance it was done under an anæsthetic. It is usually advisable that the first operation for lupus should be under an anæsthetic, because it is more

Digitized by Google

painful than one is justified in asking the patient to bear, but the later operations have required no anæsthesia. As you will see, the result has been excellent. I think the surface of the skin is really less depressed than it was before the operation, and the scar is very much less easily noticed than the disease itself was. It is two months since I last treated the case, and I now want you to carefully observe it, and you will notice, as one so often finds in dealing with tuberculosis, that there are one or two minute points of round-celled infiltration which have made their appearance —points of recurrence of growth. They become more noticable on pressure by this lens. As I have emphasized repeatedly in these post-graduate demonstrations, the success of the treatment of cutaneous tuberculosis depends on the *continuity of treatment* rather than upon any of the individual methods which you may adopt for the treatment. I mean this, that whatever means you adopt, except, perhaps, in those cases which are best suited for complete excision and skin grafting, you cannot be absolutely certain of removing the whole of the tuberculous tissues, because the tuberculous and the non-tuberculous tissues are so intimately mixed with one another that there is no means of recognising the one from the other by appearance. Therefore, the only way is to watch your cases after operation, and remove everything at once which can be removed. When we saw her at first the amount of disease was such that there was no question about the diagnosis. The recurrence of disease now is so small in amount that unless one examines it carefully it may not be noticed. If you do not destroy these recurrences, you will find in six weeks that there will be a renewal of the former condition. The secret in the treatment, whether by Finsen rays, or Röntgen rays, or by the more rapid methods of actual removal of the diseased tissue, is its *continuity*, and the careful watching for recurrences and dealing with them at once. You cannot treat your patient and then send him away to the seaside at once, unless he is under close observation. If the patient is sent to the seaside he must be watched, and any slight recurrence must be dealt with immediately. We have then in this patient a good scar produced by multiple scarification. The easiest way to remove minute nodules of recurrence in our patient is unquestionably by means of a fine point of Paquelin's cautery, or by the galvano-cautery; but perhaps this form of Paquelin's cautery is the better, as it possesses a sufficiently fine point. There is danger in the use of the actual cautery for the reason, that if you get beyond the area of disease and into the healthy tissue there is a considerable tendency to the production of hypertrophied scar, almost like keloid, which will turn your nice smooth cicatrix into one of those dense thickened scars which are so disfiguring as the result both of the disease and of its treatment. So when you come to destroy a little point of recurrence like this, be as careful in your destruction of tissue as possible; try and avoid getting beyond the margin of disease. If you do that, and destroy only the little focus of disease, then the scar which will form will be of the same nature as that which you have already produced, and you will not be likely to get these buttons of thick keloid scar which are so disappointing as results of treatment.

In answer to the gentleman who has just asked me a question, I may say that cocaine is of no use for these cases. I have tried to use most local anæsthetics, but for destruction of disease by means of a cautery they are not of much service. Anæsthesia by means of nitrous oxide is sometimes advisable, but the objections are the short duration of anæsthesia, and the movements on the patient's part. However the pain is very slight, and if you use your cautery white hot it is wonderful how well the patient stands its application. As I have told you, the first operation was performed under a general anæsthetic, but in the majority of these cases after the first attempt no anæsthetic is employed. The patient shrinks somewhat at the application, but I think that is more from the heat of the point as it approaches the face than from the puncture of the surface. My own experience in the employment of cocaine for the secondary treatment of lupus is that it is not satisfactory.

Here is a youth who has been under treatment for some time, and has been sent to me more than once for consultation. When he first came he was sent from the Metropolitan Hospital by my colleague, Mr. P. L. Daniel, as a case of *tuberculous disease*, with the request for me to say whether I thought the arm should be amputated. The condition of affairs then was very different indeed to that which you see now. The whole of the fore-arm, and part of the upper arm, was covered

Digitized by Google

by immensely swollen cutis and hypertrophied epithelium; there were great masses of cauliflower-looking overgrown epithelium over these areas. In addition, owing to the irritant secretions and the decomposition of the epithelium in the crevices between the papillary overgrowths, there was an odour given off which was almost unbearable; it was almost impossible to have him in the room. Bearing in mind that it was his right arm, and that notwithstanding this amount of disease he was still able to hold some of the tools necessary for his trade—that of a carpenter—the loss of his arm would be a very serious affair for him. I strongly dissuaded any suggestion of amputation of the arm (a course which coincided with Mr. Daniel's wish) because of the good results that have been attained by treatment. This patient has been under Mr. Daniel's care three or four months, and any of you who saw him before cannot fail to notice the great amelioration of the condition, the contrast between the state now and the original elephantoid condition of the arm is so striking. Mr. Daniel now sends him asking about the scars which are still left, and what further treatment I suggest. The course which has been pursued in the case is that the boy has been put under a general anæsthetic on various occasions, and the whole of the masses have been thoroughly well removed, and after that the arm has been dressed with applications of salicylic acid, sometimes as plaster, sometimes as paste, and sometimes as ointment, according to the surfaces to be covered, and the application has been as strong as he could bear it. That is the cause of the marvellous degree of improvement which he now presents. The question now comes as to what is the wisest thing to be done. The upper part of the scar is becoming supple; at one part the scar is very œdematous, and a good deal of infiltration still exists, but I am glad to say I also notice that the amount of round-cell infiltration in foci is very small. So that undoubtedly the prospect is good, and one may hope, under very careful treatment, looking after the young man's health thoroughly, and removing the slightest trace of recurrence as soon as it makes an appearance, and careful massage, to bring about a still greater amount of improvement. Of course in such cases as this, tuberculous lymphangitis is apt to occur, the glands enlarge, and a great deal of the trouble that takes place afterwards is not due to the recurrence of tuberculosis, but to the fact that the lymph and the venous return are obstructed; indeed, one is dealing with a case of elephantiasis as well as tuberculosis. The difficulty is to get the elephantoid tissues into good condition. Still I think you will agree with me that the suggestion made at the time—not by Mr. Daniel—that amputation should be performed, was not justifiable. He is able to take hold of some tools and use the arm fairly, and so far from being permanently disabled from earning his living, there is a prospect of still further improvement. This has been one of the worst cases of *lupus verrucosus* of the hypertrophic variety that I have ever seen.

I should like to mention one point in regard to the treatment by salicylic acid of lupus verrucosus. In this type of lupus, which often affects the extremities, we have to deal with a form of disease which tends more towards natural cure of itself than any form of tuberculosis of the skin. The reason is that in the extremities there seems to be a more than usual tendency for hypertrophy of the epithelium, owing to the inflammatory processes produced by the tubercle bacillus growing in the cutis. These masses of overgrowing epithelium not only grow upwards in the form of wart-like overgrowths, but also push downwards, and these downward processes dip into the cutis and cut off the tuberculous granulation tissue, strangling it in its actual process of growth. That is why you see in lupus verrucosus so many patches of the disease healing without treatment. If you can stimulate this process in any way, or help it, you have an opportunity of turning the balance in the right direction and helping the cure very much more easily than in cases of nodular masses of tubercular disease, such as appear in the face. If you are asked about a case of true lupus verrucosus, the prognosis of the disease is much better, and the treatment is well carried out by the means of which I have spoken—its eradication by surgical means as well as you can in the first instance, and the use of salicylic acid as strong as the patient can bear it, as a dressing subsequently. I remember one case of lupus verrucosus of the hand, in which complete cure has been obtained by means of the salicylic acid treatment alone, without any surgical procedure at all. A very good application is salicylic acid and creasote plaster. At one

Digitized by Google

time I thought the suggestion of amputation of this patient's arm was almost justified, but you will see there is no necessity for even thinking about such a thing now.

I will now show you a series of cases *not of true tuberculosis* of the skin, but cases which are believed by some to be allied to it, though perhaps not the result of the growth of the bacillus tuberculosis in the lesions. This lad has been shown at one of our meetings previously. He has been under my care for many years, indeed he has been under observation at the hospital ever since he was a little boy with what was diagnosed to be tuberculosis of the retro-peritoneal and mesenteric glands. He came under the charge of the Skin Department showing the disease known as *acne scrofulosorum*, of which you see the remains of many lesions all over the back. Let me tell you the course of the lesions as you see them even now. What happens in such cases is this: over the surface of the skin occur numerous inflammatory patches, such as these, which run the following course: small points, rarely reaching the size of a split pea, become infiltrated. The infiltration comes slowly, indeed the whole course of the lesion is a slow and chronic one, and it is made up of small round-cells, as has been proved by numerous microscopical examinations. But instead of suppurating, as one would expect from their resemblance to acne vulgaris, to your surprise you find that they do not suppurate, or only occasionally; perhaps a small amount of fluid may collect at the top of this inflammatory spot, a little exudation of serum, but no free suppuration takes place, or but very little. Of course if they become infected with septic material, such as from impetigo in another child, they are liable to suppurate. But if they run their normal course they do not suppurate but simply necrose in the centre. Perhaps a small core of material may then come out—mixed epithelium and cutis—but as a rule the necrosed area simply seems to be absorbed by the lymphatics, or rubbed off by mechanical processes. The result is that a small scar remains. This process goes on all over the body, as a rule avoiding the ordinary situations of acne vulgaris and attacking the trunk, very usually, especially the back of it, and the situations of the lesions which you see here—buttocks, thighs, and also, in the majority of cases, the upper extremities. The condition is well worthy of your attention, because it is very frequently missed unless your attention is directed to it. The relationship of this disease to tuberculosis is still doubtful. Microscopical examination in some cases has shown the tuberculous structure of the tissue, although the process is a very non-virulent one, and experimental inoculations into susceptible animals have not yet given very satisfactory results. Till recently, however, these cases have not been recognised properly, nor investigated from the histological and experimental point of view. But within the last two years a certain amount of evidence goes to prove that the lesion is an actual tuberculous one, one which is produced by the tubercle bacillus growing in the site of the lesion, although it is admitted to be of an exceedingly non-virulent type, and probably the bacilli, when present, are in very small numbers. In addition to the condition which you see in this boy, and which I will ask you to look at more carefully, he suffers from chronic corneal ulceration; he has one of those peripheral serpiginous ulcerations of the cornea, which has been going on for six or eight months, and it has been very difficult to keep it in check.

An important point in the course of the disease is the way in which the amount of infection varies from time to time. The whole eruption may almost disappear, and cases are on record in which the whole series of lesions have disappeared in a few months. Treatment by such remedies as cod-liver oil and strengthening "tonic" medicines are sufficient to turn the scale in favour of the patient; and it is not unknown that iodide of potassium in such cases has a very beneficial result. You must remember that the syphilitic granuloma is not the only granuloma which is cured by means of iodide potassium. We know that in actinomycosis the result is often most favourable, and so also in cases of tuberculosis it has sometimes a very beneficial result. Therefore do not forget that in cutaneous tuberculosis iodide of potassium is worth trying.

In this young man we have at the hospital one of the first cases, so far as I know, described in English literature, of a peculiar type of disease which seems to belong to the category of the tuberculides. The patients presents numerous small nodular elevations in the skin of the face suggesting at a little distance a violent outburst of

Digitized by Google

acne. On more careful observation you will note that there is an almost complete absence of the inflammatory erythema and œdema characteristic of acne vulgaris, and in addition that the little tumours show only here and there points of sero-purulent exudation. In the later lesions some amount of scar formation may be noted. The last thing that suggests itself is that the case is of tuberculous origin. The patient was sent to me by my colleague at Guy's Hospital, Dr. Perry, for investigation; and, as it is obviously a case in which the diagnosis is difficult, he has been taken to a meeting of the Dermatological Society of London. The type of case was recognised, but an opinion as to its exact nature was not given. To ascertain the structure of these tumours two or three of them were removed by careful excision, so as to leave as little scar as possible, and were examined microscopically. To my surprise I found that the little nodules were made up of a granulation tissue bearing the strongest resemblance to that produced by nodular cutaneous tuberculosis. Giant-cells were present of the tuberculous type, and the exudation of round-cells had also the characters of a tuberculous infiltration. A portion of the tissue was inoculated at the same time into two guinea-pigs. Unfortunately both the pigs died before it could be ascertained whether they had become tuberculous or not, so that this part of the investigation is as yet incomplete. The histological evidence is very strong in favour of the disease being a form of tuberculosis.

These cases are very rare; a very few of them have been seen, and probably described under such names as "eruption due to iodide of potassium," "acne-lupus," etc.

The treatment in this case presented much difficulty. Our patient could not be persuaded to come into the hospital, so that the use of tuberculin as a means of diagnosis was not tried, even if it were advisable. The disease was as yet non-ulcerating, producing comparatively little disfigurement, so that it was difficult to persuade the patient to have the tumours removed, and yet removal by means of fine point of the actual cautery seemed to be the proper means of treatment. Electrolysis with weak currents was tried, but produced little result.

It is remarkable to note how many obscure cutaneous diseases of doubtful origin seem to have fallen under the category of tuberculosis. We know at the present time that certain infections by other organisms simulate the tuberculous infection, so that the possibility of a disease similar to tuberculosis being produced by some other pathogenic organism than the bacillus tuberculosis must always be borne in mind; and, again, we must recollect that tuberculosis may, perhaps, be "modified," as smallpox is after vaccination.

Here I show you two patients, male and female, who have come under my care recently. Till a short time ago the disease which you see in them was considered to be one of the tuberculous manifestations. On the other hand, I think that now the distinction is better understood, and that it is pretty certain that the tubercle bacillus, at any rate *in situ*, has nothing to do with its causation. Both these patients have *lupus erythematosus*. The man shows the lesion in a very typical chronic form. He is employed in one of the wharves below London Bridge, and is exposed a good deal to the weather. About a month ago he began to suffer from the condition which you see present here, a symmetrically-distributed erythema on the cheek and neck, as a rule showing numerous white scales covering the erythematous area in the early stage of the disease, and inevitably passing on to the production of either a noticeable scar or of slighter atrophy of the skin. If you look at the patch on his left cheek you will notice that, in spite of the erythema, the surface is depressed; the reason being, no doubt, that new connective tissue, which has been formed on account of the infiltration, has developed, contraction has taken place, and, of course, the scar is produced. The extent of surface covered by the lesion in the time absolutely excludes the possibility of its being true tuberculosis. He has had it only a month, and there are multiple foci symmetrically distributed. The fact that it commenced at the age of forty-six is also strongly against the chance of it being true tuberculosis. It is rare to find true cutaneous tuberculosis starting at this time of life; it nearly always commences earlier. Remember that lupus erythematosus is very similar to some of the ordinary erythemata, *e. g.* erythema multiforme; but it is more than an erythema, infiltration takes place into the cutis, the erythema may pass off, but the disease leaves destruction of the skin.

The woman shows a rather different type of the disease. She shows lupus erythematosus affecting

Digitized by Google

the back of the hands, and affecting also both cheeks, and one other place which I will show you immediately, which is the most striking feature of the case. You see localised erythema and infiltration passing on to atrophy of the skin here, and also a wrinkling of the skin, with alteration of the little natural grooves and furrows which alter the complexion of the skin there. But it is a much more acute case than in the man; in the latter it is in the subacute or chronic form. In the woman a large area has been affected by erythema, but of this much has vanished. A much larger area on the back of the hands has been affected than you see traces of now. But in addition the woman shows lesions on the *buccal mucous membrane*, one of the rarest possible localities affected by the disease. Indeed, in most of the text-books at the present time no mention is made of the disease affecting the buccal mucous membrane. In this patient the palate is chiefly affected. It appears to be superficially excoriated and infiltrated, and slight abrasion of the surface has taken place. Perhaps the epithelium gets macerated off. I do not think the ulcers have gone through to the connective tissue. One or two small patches have appeared on the mucous membrane of the cheek in front of the anterior palatine arches.

Now just one or two remarks about these cases. As far as we know, this condition is more likely to affect the mucous membrane in the acute variety of the disease—that is to say, where it is distinguished by the greatest amount of erythema. In the exanthematous form of the disease, in which it may appear all over the body, a condition which is very dangerous to life, the mucous membrane may become affected. Therefore the more acute the case the more likely is the mucous membrane to show traces of it. In the man we see the chronic variety of the disease, and there is no trace of it in the mouth.

A word as to treatment. I have emphasised my views about the treatment of these cases so frequently that I do not think it necessary to go into it in much detail to-day. But it is quite clear I think, from the general opinions expressed, that in the treatment of lupus erythematosus you must get rid completely of the prepossessions we may have with regard to lupus verrucosus or true lupus. Anything like surgical interference would inevitably at the present time produce an exceedingly bad result. It might be easy, by means of multiple scarification, to produce a scar in the middle of this man's patch, but though you might get a scar here, or hasten the scarring process, yet all round the margin of the patch the disease would spread, and the end of it would be that you would have a larger area involved than when the treatment was started. Some years ago, before the exact knowledge of the present day was obtained, I tried that method of treatment, thinking that if anything could be done to relieve the patients at the beginning of an attack, and produce a fine superficial scar which would be little noticeable, it would be a great thing to do. I tried various other methods: scarification, curetting, and finally electrolysis, which I thought would be the most safe and gentle means of destruction of the surface. But I am sorry to say that in all the cases in which I interfered in this way I got bad results, certainly results which made me determine I would not do the same thing again. The treatment which should be carried out in lupus erythematosus, at any rate in the stage of onset, is simply the treatment which would be carried out in erythema multiforme, that is to say sedative treatment by the use of such things as oxide of zinc paste or ointment, by lotions containing calamine, oxide of zinc, lime water, glycerine, and rose water. In cases where there is itching it is desirable to add to such a preparation half or one per cent. of carbolic acid. This is a very soothing application, and the amount of carbolic acid is not sufficient to stir up the disease. By such means as these you get the best results. Internally, even in the acute stage, there is a certain amount of evidence in favour of the use of the drugs which, for want of a better term, may be called internal antiseptics. Of these, as far as we know, the best is salicin in doses of ten grains, repeated three times a day, and continued in even larger doses. I have two or three cases here under observation which have shown distinct improvement, and I have had one or two in which the disease has vanished completely under the treatment which I have just mentioned to you.

Our next patient is a woman who came up with the diagnosis of chronic eczema. In her case, however, we have not an eczema but an example of a very rare condition, which is known by various names; it is a peculiarly *atrophying folliculitis* of

Digitized by Google

the scalp, usually spreading from one focus and at length involving large areas. The erythema which accompanies the condition is often very extensive, and spreading gradually in the way that this has from the periphery, the whole of the scalp may become involved. Unfortunately, on account of the septic character of the inflammation, the production of scar tissue is great, so that permanent alopecia is almost an inevitable result. When the back of the scalp and the back of the neck are involved the condition rejoices in the name of dermatitis papillaris capillitii. I do not know of any condition affecting the back of the head of an ordinary inflammatory character capable of producing such an ugly scar. You will notice the hairs growing out of that area like small camel-hair brushes. The great point is to distinguish it from eczema. You will find the hair follicles are all inflamed and the hairs are twisted, and this is especially well seen at the margin of the lesion. This is a most inveterate condition and very resistant to treatment. Depilation does not seem to produce much effect on it, and all you can attempt to do is gradually to use some antiseptic remedy which will not irritate the skin, in the hope that it will control the septic cause, whatever it is. It appears to be allied to some of those cases which you have recognised as acne varioliformis. There are scars produced in the latter disease which can be scarcely recognised from smallpox, and apparently we have the same process going on in the scalp, producing a peculiar variety of inflammation of the follicles and atrophy of the skin. It seems to have no relationship to seborrhœa. There is some empirical evidence for the administration of sulphur here. I have used it in the form of ichthyol and thiol; of the ichthyol, five, ten, or fifteen grains should be taken daily. At the same time she is using a soap spirit lotion, which keeps the hair as well cleansed as possible; and in addition, a small quantity of resorcin and sulphur ointment,—five grains of resorcin and five grains of sulphur with paraffin.

Some of you may perhaps recognise this patient. I have him here to-day to make the diagnosis clear. When I showed him to you once before the lesion was rather difficult to make out. I told you it was a case of *erythema multiforme*, in which the eruption had passed through a well-marked iris stage, so that it could be described as a case of erythema iris. There was not much of the erythema iris at that time. But I mentioned that these cases almost inevitably recur, and it has recurred, as we expected. He has these iris lesions on both arms, and on the buccal mucous membrane. Here is a distinct wheal, almost a bulla with a round margin; it has recurred at the typical places, namely, on the extensor surfaces of the phalanges and digits, and knuckles, and extensor surfaces generally. You see it also on the lower lip. In the buccal mucous membrane it is a very trying and troublesome condition, producing great pain, salivation and difficulty in swallowing.

Various questions of importance arise in these cases, but the most interesting thing from the point of view of rarity is the possibility of the occurrence of lupus erythematosus on the mucous membranes, and the warning to be on the lookout for it, and not to pronounce it to be syphilitic glossitis or syphilitic inflammation of the palate, according to its position.

GASTRIC ULCER.

To the Editor of the CLINICAL JOURNAL.

DEAR SIR,—I notice in the CLINICAL JOURNAL of August 21st a report of a pathological demonstration by Dr. Thursfield, in which he showed a perforated gastric ulcer in a child, and states that that is "the only one he has heard of in a child under ten years."

May I call your readers' attention to a case shown by me at the Society for the Study of Disease in Children in April, 1901, when I showed a specimen of a gastric ulcer in a child aged two years, which had caused death by perforation and subsequent peritonitis? Three cases have also been recorded by Dr. Soltau Fenwick from the records of the London Hospital.

I am, Sir, very truly yours,

J. PORTER PARKINSON.

Wimpole Street;
September 30th, 1901.

Digitized by Google

INDEX TO VOLUME XVIII.

PAGE

Abraham, Phineas S., demonstration of cases of skin diseases 392
Allbutt, T. Clifford, "some neuroses" . . 225
Amenorrhœa, ergot and strychnine in . . 213
Anæsthetics, the after effects of, by J. Blumfeld 252
Aneurysm, the treatment of, by W. McAdam Eccles 348, 363
Ankle, case of old injury to, by James Berry 281
Arsenic and erythromelalgia 144
Arsenical poisoning from beer drinking, by T. N. Kelynack 222
Arterial hæmorrhage, the control of, by W. McAdam Eccles 348, 363
Arthritis, mechanical or traumatic, by W. Arbuthnot Lane . . 358, 377
" rheumatoid, toxic origin of, by N. C. Macnamara 44
Atrophy, muscular, by J. H. Bryant . . 327

Bacteriology of urine 224
Ballance, C. A., removal of a hat-pin from the stomach of an infant 64
Bannatyne, Gilbert A., the relationship between acute infective rheumatism and rheumatoid arthritis 53
Barlow's disease, case of 187
Batten, F. E., cerebral diplegia in children . 33
Beaumont, W. M., four cases of intra-ocular hæmorrhage 335
Beer drinking, arsenical poisoning from, by T. N. Kelynack 222
Beevor, Sir Hugh, two cases of general venous engorgement 14
Bennett, Sir William, certain complications occurring after operations for the cure of hernia . 241
" points connected with thrombosis . . 401
Berry, James, at the Medical Graduates' College and Polyclinic 782

PAGE

Bloodletting, practice of, by J. F. Briscoe 76, 90, 174, 299
Blumfeld, J., the after effects of anæsthetics . 252
Bone grafting 352
Boric acid or borax in the feeding of infants 235
Boyd, Stanley, at the Hospital for Consumption and Diseases of the Chest, Brompton . 145
Bright's disease, ophthalmoscopic changes met with in, by Percy Flemming . . 109
Briscoe, J. F., practice of bloodletting . 76, 90, 174, 299
Bruce, J. Mitchell, the diagnosis, prognosis, and treatment of quiescent tuberculosis . 209
Brunton, Sir Lauder, the medicinal treatment of disease 6
Bryant, J. H., causation and diagnosis of acute intestinal obstruction . 113, 136
" muscular atrophy . . . 327
Butlin, H. T., the surgical treatment of tuberculous glandular affections in the neck . 321

Cancer of tongue, treatment of . . . 272
Carcinoma, extensive, of bladder and rectum, case of, by James Berry 283
Cardiac pain and its treatment, by Arthur Latham 396
"Case-taking," practical outline of, by Frederick Roberts 257
Cerebral diplegia in children, by F. E. Batten 33
" rheumatism or chorea, by Sir Dyce Duckworth 236
Cerebro-spinal meningitis and otitis . . 26
Chelsea Clinical Society, discussion by the 1, 17, 38, 53
Cheyne, W. Watson, at King's College Hospital 49
Chloretone in dusting powders . . . 151
Cholelithiasis 13
Chorea, the incidence, nature, and treatment of, by Sir Dyce Duckworth . . 369
" or cerebral rheumatism, by Sir Dyce Duckworth 236

Digitized by Google

PAGE
Chorea of pregnancy, by Amand Routh . 193
Circumcision, case of, by H. Stansfield Collier 317
Colds, physiological care of 75
Collier, H. Stansfield, at the Hospital for Sick Children 315
Coloboma of each iris, case of, by C. O. Hawthorne 221
Coxa vara, case of, by W. Watson Cheyne . 49
Creosotal in scrofulous affections and ozæna . 80
Creosote in tuberculosis 272
Cyst, diagnosis of an ovarian, by Amand Routh 65
Cysts, dermoid, in a child 320

Dermoid cysts in a child 320
Diagnosis, cardinal rules in, by C. B. Lockwood 172
Diarrhœa of infants, by J. D. Windle . . 191
Digitalis 265
Dionin in disease of the cornea . . . 256
Diplegia, cerebral, in children, by F. E. Batten 33
Duckworth, Sir Dyce, chorea or cerebral rheumatism . . . 236
,, clinical and pathological relations of the chronic rheumatic and rheumatoid affections to acute infective rheumatism . 23
,, incidence, nature, and treatment of chorea . 369
Dupuytren's contraction, case of, by W. Watson Cheyne 50
Dysentery, South African, by J. W. Washbourn 289
Dyspnœa caused by goitre 96

Ear, living animal organisms in the . . 172
Eccles, W. McAdam, the control of arterial hæmorrhage and the treatment of aneurysm 348, 363
Eden, T. W., gonorrhœa in women . . 342
Electric current of high tension, action of, on the human body 294
Engorgement, general venous, two cases of, by Sir Hugh Beevor 14
Epididymis, case of enlargement of the, by Jonathan Hutchinson 128
Epithelioma of soft palate and tongue, cases of, by James Berry 283, 284
Errors in laboratory diagnosis . . . 122

PAGE
Erythromelalgia and arsenic 144
Eyelid, swelling of the, by Theodore Fisher . 318

Fever, yellow, and snake-bite, analogy between 284
Fisher, Theodore, two cases of swelling of the eyelid occurring after a bath . . . 318
Fistulæ, vesico-vaginal, operation for . . 111
Flat-foot, case of, by W. Watson Cheyne . 51
Flemming, Percy, ophthalmoscopic changes met with in Bright's disease . . . 109
Formaldehyde 190
Fracture dislocation in upper dorsal region, recovery from, by Charles A. Morton 372
,, of forearm with paralysis of ulnar nerve, case of, by James Berry . 279
,, old, near ankle, case of, by James Berry 280
Fractures, treatment of 256
Freyer, P. J., at the Medical Graduates' College and Polyclinic 177

Galloway, J., in the Skin Department of Charing Cross Hospital 410
Garrod, A. E., rheumatism and rheumatoid arthritis 39
Glanders, case of acute, by W. C. C. Pakes . 112
Goitre, dyspnœa caused by 96
Gonorrhœa in women, by T. W. Eden . . 342
Grafting, bone 352

Hæmorrhage, intra-ocular, by W. M. Beaumont 335
,, arterial, the control of, by W. McAdam Eccles . . 348, 363
Hat-pin, removal of, from the stomach of an infant, by C. A. Ballance 64
Hawthorne, C. O., at the Medical Graduates' College and Polyclinic . 214
,, relationsnip of acute rheumatism to chronic rheumatoid arthritis . . 46
Head, blows on the 221
Hensley, P. J., clinical lecture . . . 353
Hernia, certain complications occurring after operations for the cure of, by Sir William Bennett 241
Heroin, a bronchial sedative. 192
Hutchinson, Jonathan, at the Medical Graduates' College and Polyclinic 15, 74, 126

Digitized by Google

PAGE
Hutchinson, Jonathan, Raynaud's phenomena 305
Hydrocele, by A. Marmaduke Sheild . . 129

Intestinal obstruction, causation and diagnosis of acute, by J. H. Bryant . 113, 136
,, ulceration, by C. B. Keetley . 247, 263
Intussusception and its treatment, by D'Arcy Power 268
Iron, technique of subcutaneous injections of 351

Jaundice due to cancer of the common bile-duct, case of, by James Berry . . . 278

Keetley, C. B., intestinal ulceration . 247, 265
Kellock, Thomas H., nævi and their treatment 17
Kelynack, T. N., arsenical poisoning from beer drinking 222
Kidney, movable, by W. Arbuthnot Lane . 97
,, ,, case of, by W. Watson Cheyne . . . 52
,, some affections of the, by A. H. Tubby . . 86, 122, 157, 187
,, suspected tuberculous, case of, by P. J. Freyer 179

Lane, W. Arbuthnot, mechanical or traumatic arthritis . . 358, 377
,, movable kidney . . 97
,, treatment of foul wounds 199
Latham, Arthur, cardiac pain and its treatment 396
Lees, D. B., at the Hospital for Sick Children 385
Lichen planus treated as a syphilide with mercury 126
,, spinulosus, case of, by Mr. Jonathan Hutchinson 126
Lockwood, C. B., cardinal rules in diagnosis 172

Macnamara, N. C., toxic origin of rheumatoid arthritis 44
Mechanical or traumatic arthritis, by W. Arbuthnot Lane 358, 377
Medicinal treatment of disease, by Sir Lauder Brunton 6
Meningitis, cerebro-spinal, and otitis . . 26
Mercury, lichen planus treated with . . 126
Morton, Charles A., recovery from fracture dislocation in the upper dorsal region . 372
Moullin, C. Mansell, treatment of thrombosed varicose veins of the leg 239
Muscular atrophy, by J. H. Bryant . . 327

PAGE
Nævi and their treatment, by Thomas H. Kellock 17
"Neuroses, some," by T. Clifford Allbutt . 225

Ophthalmoscopic changes in Bright's disease, by Percy Flemming 109
Optic atrophy, double, case of, by C. O. Hawthorne 214
Osteo-arthritis, on the diagnostic value of X-ray photography in, by E. W. H. Shenton 85
Otitis and cerebro-spinal meningitis . . 26
Ovarian cyst, diagnosis of an, by Amand Routh 65

Paine, Alexander, infectivity of acute rheumatism 41
Pakes, W. C. C., case of acute glanders . 112
Paraffin protheses, Gersuny's subcutaneous . 299
Paralysis, pseudo-hypertrophic, case of, by C. O. Hawthorne 218
Peritonitis, tubercular, by W. Hale White . 273
Picric acid in smallpox 347
Pitts, Bernard, at the Hospital for Sick Children 405
Pleurisy and quiescent tuberculosis, by J. Mitchell Bruce 212
Poisoning, arsenical, from beer drinking, by T. N. Kelynack 222
Power, D'Arcy, intussusception and its treatment 268
Poynton, F. J., infectivity of acute rheumatism 41
Pregnancy, the chorea of, by Amand Routh . 193
Prostate, total extirpation of the, by P. J. Freyer 177
Puerperal fever, treatment of . . . 157
Pupil after death 70

Quinine, idiosyncrasy to 108

Raynaud's phenomena, by Jonathan Hutchinson 305
Reflex, plantar, in infancy 15
Rheumatism, acute, infectivity of, by F. J. Poynton and Alexander Paine . . 41
,, ,, relationship to chronic rheumatoid arthritis, by C. O. Hawthorne 46

Digitized by Google

PAGE

Rheumatism, acute infective, and rheumatoid arthritis, on the relationship between, by G. A. Bannatyne . 53
„ clinical and pathological relations of the chronic rheumatoid affections to acute infective, by Sir Dyce Duckworth 1, 23
„ rheumatoid arthritis, and subcutaneous nodules . . . 336
Rheumatoid arthritis, toxic origin of, by N. C. Macnamara 44
Roberts, Frederick, practical outline of "case-taking" 257
Roughton, E. W., at the Medical Graduates' College and Polyclinic 294
Routh, Amand, chorea of pregnancy . . 193
„ diagnosis of an ovarian cyst . 65

Sarcomatous tumour, case of multiple subcutaneous, by Jonathan Hutchinson . . 74
Scirrhus of the breast, case of inoperable, by W. Watson Cheyne . . 51
„ male breast, case of, by James Berry 282
Seminal vesiculitis, chronic 203
Sheild, A. Marmaduke, hydrocele . . 129
Shenton, E. W. H., the diagnostic value of X-ray photography in osteo-arthritis . . 85
Skiagraphy, by Hugh Walsham 152, 161, 184, 203
Skin diseases, cases of, by J. Galloway . 410
„ demonstration of cases of, by Phineas S. Abraham . . 392
Smallpox, picric acid in 347
Smith, E. Noble, spring finger . . . 31
„ Priestley, strabismus 337
Snake-bite, yellow fever and . . . 284
Spinal caries, case of, by James Berry . . 282
Spring finger, by E. Noble Smith . . . 31
Strabismus, by Priestley Smith . . . 337
Strangulation, anatomic conditions in . . 277
Supra-renal extract, local action on nose and throat 198
Syers, H. W., clinical lecture . . . 27

Tabes, case of, by Jonathan Hutchinson . 128
„ dorsalis, by W. Aldren Turner . . 71
Talipes, congenital, cases of, by H. Stansfield Collier 315

PAGE

Thrombosed varicose veins of leg, treatment of, by C. Mansell Moullin 239
Thrombosis, by Sir William Bennett . . 401
Thursfield, J. H., pathological demonstration 284
Tongue, treatment of cancer of . . . 272
Traumatic or mechanical arthritis, by W. Arbuthnot Lane 358, 377
Tubby, A. H., some affections of the kidney 86, 122, 157, 187
Tubercular peritonitis, by W. Hale White . 273
Tuberculosis, creosote in 272
„ diagnosis, prognosis, and treatment of quiescent, by J. Mitchell Bruce . . . 209
Tuberculous glandular affections in the neck, surgical treatment of, by H. T. Butlin . 321
Turner, W. Aldren, tabes dorsalis . . . 71
Typhoid fever, modified treatment of . . 183

Ulcer, chronic, of the leg, case of, by James Berry 281
Ulceration, intestinal, by C. B. Keetley 247, 265
Urates, white, specimen of, by C. O. Hawthorne 221
Urine, bacteriology of the 224
Uterus, retroflexion and retroversion of the gravid 174

Varicose veins of the leg, treatment of thrombosed, by C. Mansell Moullin . . . 239
Venous engorgement, general, two cases of, by Sir Hugh Beevor 14
Vesiculitis, chronic seminal 203

Walsham, Hugh, present-day skiagraphy . 152, 161, 184, 203
Washbourn, J. W., South African dysentery 289
White, W. Hale, in the wards of Guy's Hospital . . . 81, 104
„ tubercular peritonitis . . 273
Windle, J. D., summer diarrhœa of infants . 191
Wounds, foul, treatment of, by W. Arbuthnot Lane 199
Wry-neck, diagnosis in 144

X-ray photography, on the diagnostic value of, in osteo-arthritis, by E. W. H. Shenton 85

Yellow fever and snake-bite . . . 284

LONDON: PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE, E.C.; 20 HANOVER SQUARE, W.; AND DORKING.

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

51

22454

Digitized by Google